Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING

THE INDEX OF OTOLARYNGOLOGY

L. W. DEAN, M. D., Editor-in-Chief
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TABLE OF CONTENTS.

I.—Report of the Ear and Throat Department, Royal Infirmary, Edinburgh (Year 1927). Under the Charge of J. S. Fraser, M. B., F. R. S. C. Ed. Statistical Tables and Case Records by G. L. Reid, M. B., Ch. B. Edin..... 1

II.—The Treatment of Cancer of the Pharynx, Larynx and Esophagus by Surgical Diathermy. By Dan Mackenzie, M. D., London, Eng..... 32

III.—Vasomotor Affections of the Internal Ear. By Professor Georges Portmann, M. D., Bordeaux..... 69

IV.—Headache From the Standpoint of the Rhinologist. By Henry Dintenfass, M. D., Philadelphia..... 77

V.—Diagnosis of Antral Infection. By Matthew S. Ersner, M. D., Philadelphia..... 87

VI.—X-Ray Examination of Deformities of the Nasopharynx. By Prof. Arthur Schüller, M. D., Vienna, Austria..109

VII.—The Development of the Sinuses After Birth. By T. E. Carmody, M. D., Denver.....130

VIII.—Some Practical Considerations in the Problem of Sinus Infection and Drainage. By Harry L. Baum, M. D., Denver 152

IX.—Nitrous-Oxid-Oxygen Anesthesia for Tonsillectomies. By John H. Evans, M. D., Buffalo, N. Y.....158

X.—Colonic Anesthesia in Operations of Otolaryngologic Cases. By Edward W. Beach, M. D., Philadelphia.....169

XI.—Response of Vestibular Apparatus to Drugs. By Ellison L. Ross, M. D., and Murland W. Fish, M. D.....175

XII.—Otitis Media and Allergy. By Eugene R. Lewis, M. D., Los Angeles.....185

XIII.—Vertigo as a Symptom. By H. Moulton, M. D., Fort Smith, Ark.....189

XIV.—Some Very Accurate Measurements of the Amplitude of Vibration of Tuning Fork Prongs. By Robert Sonnenschein, M. D., Chicago.....197

XV.—Anatomy, Physiology and Pathology of the Lacrimal Apparatus, More Especially the Lacrimal Sac and Duct. By Harry Vanderbilt Würdemann, M. D., Seattle.....199

XVI.—Retropharyngeal Abscess. By Roy A. Barlow, M. D., Madison, Wis. 205

XVII.—Complications of Otitis Media and Mastoiditis. By Edward H. Campbell, M. D., Philadelphia 209

XVIII.—Infantile Mastoiditis With Gastrointestinal Symptoms: Report of Two Cases. By James B. Costen, M. D., St. Louis 242

XIX.—Noncoalescent Mastoiditis With a Report of Four Cases. By Sam B. Marks, M. D., Lexington, Ky. 251

XX.—The Nature of Nystagmus and Dizziness Occurring in Certain Positions of the Head and in Movements Other Than Those Around the Long Axis of the Body. By Ignatz Sommer, M. D., Vienna, Austria, and Joseph C. Yaskin, M. D., Philadelphia 260

XXI.—A Positive Symptom of Mastoiditis. By Murray Berger, M. D., New York 275

XXII.—Chronic Laryngeal Stenosis Complicating Laryngeal Diphtheria. By R. J. Payne, M. D., St. Louis 276

XXIII.—Toxic Otitis. By J. J. Hompes, M. D., Lincoln, Nebraska 280

XXIV.—Unilateral Blindness: Recovery After Sinus Operation. By J. T. Dowling, M. D., Seattle 288

XXV.—Anaphylaxis and Allergy in Rhinology. By Francis P. Emerson, M. D., Boston 313

XXVI.—The Coincidence of Meningeal Calcification and Nasal Pathology. By James A. Babbitt, M. D., Philadelphia, Pa. 320

XXVII.—Brain Abscess and Meningitis Secondary to Acute Paranasal Sinusitis. By John B. Potts, M. D., Omaha 329

XXVIII.—Trigeminal Disturbances of Otitic Origin. By French K. Hansel, M. D., St. Louis 335

XXIX.—Perforation of the Esophagus, with Report of Six Cases. By Edward King, M. D., Cincinnati 351

XXX.—Histopathology of Nose and Throat in Experimental Cretinism. By Samuel J. Pearlman, M. D., Myron E. Kahn, M. D., and M. M. Kunde, Ph. D., M. D., Chicago 363

XXXI.—Nerve Block in Amygdalectomy. By Homer A. Trotter, M. D., Buffalo 376

XXXII.—Abortive Treatment of Peritonsillar Inflammation. By W. R. THOMPSON, M. D., Fort Worth, Texas.....386
XXXIII.—Encephaloma or the So-called Nasal Glioma. By JEFFERSON BROWDER, M. D., Brooklyn.....395
XXXIV.—Pathogenesis of Osteomas of the Nasal Accessory Sinuses. By ALEXANDER G. FETISSOF, M. D., Tomsk, Siberia 404
XXXV.—Value of Barbital Before Local Anesthesia. By ROY A. BARLOW, M. D., Rochester, N. Y.....421
XXXVI.—The Incidence of Ear, Nose and Throat Defects in Lincoln School Children. By EARL B. BROOKS, M. D., Lincoln, Neb.....424
XXXVII.—Treatment of Dacryocystitis from the Viewpoint of the Rhinologist. By ARTHUR C. JONES, M. D., Boise, Idaho 435
XXXVIII.—Otologic Complications from Swimming in Summer Camps: An Attempt at Prevention. By FREDERICK T. HILL, M. D., Waterville, Maine.....444
XXXIX.—Some Unusual Cases of Lateral Sinus Thrombosis. By ARTHUR M. ALDEN, A. M., M. D., St. Louis.....455
XL.—The Mastoids, Foci of Infection for Arthritis: Report of a Case. By TAYLOR S. BURGESS, M. D., Atlanta, Ga.....465
XLI.—Agranulocytic Angina: Report of Two Cases, One Death and One Recovery. By R. E. WINDHAM, M. D., Longview, Wash.....470
XLII.—Ethmoiditis Producing Hemiplegia, Concommitant with Toxic Scarlet Fever: Operation, Cure. By MORRIS A. WEINSTEIN, M. D., Philadelphia.....478
XLIII.—Otitic Brain Abscess. By FREDERICK A. KIEHLE, A. B., M. D., Portland, Ore.....482
XLIV.—An Otolaryngologic Reading Club. By JESSE H. ROTH, M. D., and SAMUEL J. PEARLMAN, M. D., Chicago....488
XLV.—President's Address. By CHARLES W. RICHARDSON, M. D., Sc. D., Washington.....493
XLVI.—Sinusitis in Children. By J. MACKENZIE BROWN, M. D., Los Angeles.....500
XLVII.—An Analysis of Five Hundred Tonsillectomies. By M. C. COMER, M. D., Tucson, Arizona.....506
XLVIII.—GHERARDO FERRERI. (November 15, 1856—January 22, 1929).....516

XLIX.—Vitamins in Human and Animal Nutrition. By Professor R. Adams Dutcher, State College, Pa.....593

L.—Diet in Connection With Diseases of the Lymphatic System, Especially of the Upper Air Tract. By Roy A. Barlow, M. D., Rochester, N. Y.....600

LI.—The Relation of Deficiency Diet to Diseases of the Sinuses. By Lee Wallace Dean, M. D., St. Louis.....607

LII.—Deficiency Diet in Relation to the Skeleton, Especially in Connection With the Bone Affections of the Head. By B. R. Shurly, M. D., Detroit, Mich.....612

LIII.—The Necessity for a Scientific Investigation of the Causes of Hypertrophy of the Lymphoid Elements of the Pharynx. By D. Bryson Delavan, M. D., New York.....622

LIV.—Deafness in Syphilis: An Audiometric Study. By Dana W. Drury, M. D., Boston.....625

LV.—Further Experience With the Dichloramin Treatment of Mastoid Wounds. By Ralph A. Fenton, M. D., Portland, Oregon 657

LVI.—A Contribution to the Study of Chronic Progressive Deafness, With a Plea for a Nation-Wide Investigation. By Thomas Jefferson Harris, M. D., New York.....661

LVII.—A Case of Infection by Brucella Melitensis Var. Abortus Complicating Tonsillectomy. By George Fetterolf, M. D., Sc. D., Philadelphia.....675

LVIII.—The Sinus in Perspective. By Arthur W. Proetz, M. D., St. Louis.....682

LIX.—Considerations for and Against Curetting and Exenterating Sinus Operations. By Eugene R. Lewis, M. D., Los Angeles 685

LX.—Specific Protein Reactions in Eye, Ear, Nose and Throat. By George Piness, M. D., and Hyman Miller, M. D., Los Angeles.....691

LXI.—Report of a Case of Mixed Tumor of the Parotid Type Growing From the Posterior Aspect of the Thyroid Cartilage. By R. C. Lynch, M. D., New Orleans.....706

LXII.—Chronic Ethmoiditis: Its Conservative Surgical Treatment. By Ross Hall Skillern, M. D., Philadelphia..716

LXIII.—Histopathology of Sinusitis and Mastoiditis. By Bernard J. McMahon, M. D., M. Sc. (Med.), St. Louis....732

LXIV.—The Relation of Sinus Disease to Hay Fever. By T. E. CARMODY, M. D., and L. W. GREENE, M. D., Denver..759

LXV.—Limitations of the Functional Tests in the Differential Diagnosis of Deafness. By EDMUND PRINCE FOWLER, New York 766

LXVI.—Anatomic Studies of the Sphenopalatine Ganglion and the Posterior Palatine Canal, With Special Reference to the Use of the Latter as the Injection Route of Choice. By EDWARD H. CAMPBELL, M. D., Philadelphia..... 778

LXVII.—Chronic Nonpurulent Sinusitis and Its Clinical Significance. By FRANK B. KISTNER, M. D., Portland, Oregon 795

LXVIII.—The Use of the New Magnesium Alloy Tuning Forks. By ROBERT SONNENSCHEIN, M. D., Chicago..... 805

LXIX.—Nontuberculous Lesions of the Larynx in the Tuberculous. By JOSEPH B. GREENE, M. D., Asheville, North Carolina 812

LXX.—Education of the General Physician and Public to the Sinus Problem. By H. B. LEMERE, M. D., Omaha..... 820

LXXI.—The Function of the Muscular Attachments of the Tonsil. By HOMER A. TROTTER, M. D., Buffalo..... 825

SYMPOSIUM ON THE ETHMOID

LXXII.—The Surgical Anatomy of the Ethmoidal Labyrinth. By HARRIS P. MOSHER, M. D., Boston..... 869

LXXIII.—Pathology and Diagnosis. By ROSS HALL SKILLERN, M. D., Philadelphia..... 902

LXXIV.—Conservative Treatment of Ethmoiditis. By SAMUEL IGLAUER, M. D., Cincinnati..... 908

LXXV.—Radical Treatment of the Ethmoid: Intranasal. By RALPH A. FENTON, M. D., Portland, Ore..... 913

LXXVI.—Symposium on Ethmoidal Sinusitis: External Radical Approach. By R. C. LYNCH, M. D., New Orleans 931

LXXVII.—Heredity and Human Biology. By G. H. PARKER, Professor of Zoölogy, Harvard University, Cambridge..... 940

LXXVIII.—Recent Advances in Otolaryngology: Medical and Surgical Treatment. By LEE WALLACE DEAN, M. D., St. Louis 952

SYMPOSIUM ON THE SINUSES

LXXIX.—Physics and Physiology of Sinuses. By ARTHUR W. PROETZ, M. D., St. Louis....963

LXXX.—Pathology of Sinusitis. By FRANK B. KISTNER, M. D., Portland, Ore....977

LXXXI.—General Surgical Principles Employed in the Treatment of Chronic Disease of the Nasal Sinuses. By EDWARD CECIL SEWALL, M. D., San Francisco....995

LXXXII.—General Principles of Treatment of Nasal Sinus Inflammations. By EUGENE R. LEWIS, M. D., Los Angeles....1005

LXXXIII.—An Extensive Case of Acute Osteomyelitis of the Frontal and Superior Maxillary Bones Complicating Sinusitis, Operation, Apparent Recovery, Sudden Death. By ROSS HALL SKILLERN, M. D., Philadelphia....1022

LXXXIV.—The Occurrence of Tympanic Hemorrhage Following the Radical Operation for the Relief of Trigeminal Neuralgia. By W. J. GARDNER, M. D., and JAMES A. BABBITT, M. D., Philadelphia....1040

LXXXV.—Iodized Oil as an Aid in the Diagnosis of Sinus Disease. (Campiodol-Lipiodol.) By KARL MUSSER HOUSER, Ph. B., M. D., M. Sc., Philadelphia....1052

LXXXVI.—Iodized Rapeseed Oil (Campiodol) in the Diagnosis of Chronic Maxillary Sinusitis. By MARK A. GLASER, M. D.; CHARLES E. FUTCH, M. D., and HENRY SNURE, M. D., Los Angeles....1067

LXXXVII.—Asthma: Its Etiology and Surgical Treatment. By FERRIS SMITH, A. B., M. D., F. A. C. S., Grand Rapids Clinic, Grand Rapids....1095

LXXXVIII.—An Analysis of the Limb Responses to Semicircular Canal Stimulation in the Frog. By JOHN TAIT, M. D., and W. J. MCNALLY, M. D., Montreal....1121

LXXXIX.—The Use of Electrically Heated Bougies in the Treatment of Postdiphtheritic Cicatricial Stenosis of the Larynx. By W. H. JOHNSTON, M. D., St. Louis....1145

XC.—Nonsurgical Treatment of Chronic Middle Ear Suppuration, Based Upon Twenty-five Years' Experience. By JOSEPH C. BECK, M. D., Chicago....1150

XCI.—Method of Correcting Lax, Baggy Septa Following Submucous Resection. By JOHN A. PRATT, M. D., Minneapolis 1156

XCII.—Two Interesting Foreign Body Cases of the Mastoid and the Nose. By EDWIN P. LONGAKER, M. D., Philadelphia 1158

XCIII.—Five Lectures on the Physiology of the Ear. By W. J. MCNALLY, M. D., Montreal.

LECTURE 1.—The Physiology of the Cochlea With Some Remarks About the Clinical Tests for Hearing 1163

LECTURE 2.—The Physiology of the Saccule and Saccus Endolymphaticus 1180

EDITORIAL.—The American Board of Otolaryngology 1197

XCIV.—Dedication of Central Institute for the Deaf. By L. W. DEAN, M. D., St. Louis 1199

Abstracts of Current Articles 297, 519, 833, 1202

Books Received 298

Society Proceedings 301, 523, 839

American Board of Otolaryngology 836

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III.—Vasomotor Affections of the Internal Ear. By Professor George Portmann, M. D., Bordeaux.....69

IV.—Headache From the Standpoint of the Rhinologist. By Henry Dintenfass, M. D., Philadelphia.....77

V.—Diagnosis of Antral Infection. By Matthew S. Ersner, M. D., Philadelphia 87

VI.—X-Ray Examination of Deformities of the Nasopharynx. By Prof. Arthur Schüller, M. D., Vienna, Austria.....109

VII.—The Development of the Sinuses After Birth. By T. E. Carmody, M. D., Denver.....130

VIII.—Some Practical Considerations in the Problem of Sinus Infection and Drainage. By Harry L. Baum, M. D., Denver.....152

IX.—Nitrous-Oxid-Oxygen Anesthesia for Tonsillectomies. By John H. Evans, M. D., Buffalo, N. Y.....158

X.—Colonic Anesthesia in Operations of Otolaryngologic Cases. Edward W. Beach, M. D., Philadelphia.....169

XI.—Response of Vestibular Apparatus to Drugs. By Ellison L. Ross, M. D., and Murland W. Fish, M. D.....175

XII.—Otitis Media and Allergy. By Eugene R. Lewis, M. D., Los Angeles.....185

XIII.—Vertigo as a Symptom. By H. Moulton, M. D., Fort Smith, Ark. 189

XIV.—Some Very Accurate Measurements of the Amplitude of Vibration of Tuning Fork Prongs. By Robert Sonnenschein, M. D., Chicago 197

CONTENTS—Continued on Third Cover Page.

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VOL. XXXVIII. MARCH, 1929. No. 1.

I.

REPORT OF THE EAR AND THROAT DEPARTMENT, ROYAL INFIRMARY, EDINBURGH (YEAR 1927).

UNDER THE CHARGE OF J. S. FRASER, M. B., F. R. S. C. ED.
STATISTICAL TABLES AND CASE RECORDS BY
G. L. REID, M. B., CH. B. EDIN.

The following report gives an account of the work done during the year 1927 in the section of the Ear and Throat Department of the Royal Infirmary under my charge. The cases are grouped according to the region or regions affected —e. g., nose, paranasal sinuses, pharynx, larynx and ear— and, as far as possible, according to the following classification —e. g., congenital and traumatic affections, acute and chronic inflammations, tumors and nervous affections. The number of diseases exceeds the number of patients seen because if a patient suffered from—e. g., adenoids and chronic middle ear suppuration—he appears under both headings.

The various operations performed have also been tabulated. It will be noted that the cases and operations do not always correspond; this is due to the fact that many of the operations in the earlier part of the year were performed on patients who

had been seen in 1926. A more or less detailed account is given of certain interesting and successful cases and also of all fatal cases. Most of our tonsil and adenoid operations on children (Whillis-Sluder method) are not kept in the wards but are sent home in taxi cabs about four hours after operation. Some of the nasal septum cases have also to be dealt with in the same way, owing to the lack of beds and the length of the waiting list. Further, we have not in Edinburgh any efficient "follow-up" system, so that in these two respects the report is admittedly incomplete.

Dr. Reid has, however, given as thorough and candid a report as is possible under the circumstances. There can be little doubt that if similar reports were compiled and published in the various hospitals and departments in the United States, where all cases are kept in hospital after operation, and where there is an efficient "follow-up" system, such reports would be of the greatest value in ascertaining the incidence of the various diseases, the results of treatment and the mortality associated with the various operative procedures.

Affections of the Nose (2045).

I. THE EXTERNAL NOSE AND NASAL VESTIBULES.

Collapse of alae nasi	4
Injury to nose, including fracture of nasal bones	30
Dermatitis of external nose and nasal vestibule	42
Furuncle of nasal vestibule	8
Erysipelas of nose	2
Lupus of external nose	1
Cyst of anterior nares	2
Papilloma of nasal vestibule	3
Rhinophyma	1
	93

II. THE NASAL CAVITIES.

Deflection of septum to right	356
Deflection of septum to left	339
Irregular deflections of septum	219
Foreign bodies in nose	6
Epistaxis	49

Adhesions in nasal cavities	10
Acute, subacute and chronic rhinitis	154
Rhinitis sicca	7
Simple perforation of septum	15
Simple ulcer of septum	1
Hematoma and abscess of septum	2
Purulent rhinitis	13
Atrophic rhinitis (nonfetid)	14
Atrophic rhinitis (fetid) (ozena)	23
Inferior turbinal enlargement	382
Polypoid middle turbinals and nasal polypi	143
Rhinolith	2
Tuberculoma of septum	1
Lupus of nasal mucous membrane	2
Syphilitic (tertiary) disease of nasal cavities	6
Hemangioma of septum	2
*Fibrosarcoma of inferior turbinal	1
Nasal neuroses (including asthma)	104
Sphenopalatine ganglion syndrome	1
	1952

OPERATIONS ON THE NOSE.

Foreign body removed (general anesthesia)	4
Nose straightened	13
Abscess of septum opened	1
Submucous resection of nasal septum	131
Submucous resection of nasal septum and inferior turbinotomy	96
Submucous resection of nasal septum and polypi	5
Nasal polypi (including intranasal ethmoid and Sluder's operation)	31
Inferior turbinotomy	62
Inferior turbinotomy (posterior ends)	9
Middle turbinotomy (excluding intranasal ethmoid and Sluder's operation)	12
Inferior and middle turbinotomy	1
Plastic operation (one rhinophyma)	2
Simple tumor (hemangioma) removed	1

*Case recorded.

Rhinolith removed	2
Fibrosarcoma removed	1
Cautery removal of simple tumor	3
Diathermy (lupus 3, nasopharyngeal fibroma 1)	4
Radium inserted into nose	1
Injection of patient's own blood into inferior turbinals (for ozena)	1
	380

No deaths from nasal operations.

FIBROSARCOMA OF LEFT INFERIOR TURBINAL.

J. McA., male, aged 38 years. First seen February 28, 1927, complaining of nasal obstruction, especially on left side, for two months; profuse thick, crusty nasal discharge, with bad odor; frequent frontal headaches.

On anterior rhinoscopy a large purple swelling is seen attached to the left inferior turbinal. Patient says he removed some of this six weeks ago and that it bled profusely.

May 16, 1927. Soft swelling removed under local anesthesia, with snare, and sent for microscopic examination. The pathologist reported "Growth consists of whorls of spindle-shaped cells showing variations in size and shape; there are a good many mitotic figures indicating active growth. On the other hand, the vessels of which there are many, have fairly well formed walls. There is no definite evidence of malignancy but the appearances are rather suggestive of a fibrosarcoma."

THE PARANASAL SINUSES (125).

Acute antral catarrh	5
Chronic antral catarrh	10
Acute antral suppuration	11
Chronic antral suppuration	29
Acute frontal sinus catarrh	15
Acute frontal sinus suppuration	12
Acute ethmoiditis	2
Chronic ethmoiditis (catarrhal and suppurative)	11
Acute sphenoid sinusitis	2
Chronic sphenoid sinus suppuration	2

Acute and chronic antral and ethmoid suppuration	6
Acute antral and frontal suppuration	1
Chronic ethmoid and sphenoid suppuration	3
Chronic antral, ethmoid and sphenoid suppuration	1
Acute antral, ethmoid and frontal catarrh	1
Chronic antral, ethmoid and frontal suppuration	2
Pansinusitis	1
Dental cyst invading antrum	5
Antro-naso-choanal polypi	5
Pneumatocele or mucocele of frontal sinus	1
	125

Dacryocystitis	6

OPERATIONS ON THE PARANASAL SINUSES (INCLUDING WEST'S TEAR SAC OPERATION).

Intranasal antrum	13
Radical antrum (Caldwell-Luc) (one death)	30
Antro-naso-choanal polypus	5
Dental cyst invading antrum	5
Intranasal frontal	5
Intranasal ethmoid and sphenoid (including Sluder's operation)	38
	96

West's tear sac operation	3

I. FATAL CASE FOLLOWING RADICAL ANTRUM OPERATION.

A. C., female, aged 33 years, complained of nasal obstruction for two months, with frontal headache and bad smell (subjective).

Examination showed pus in left middle meatus; proof puncture of left antrum positive.

Caldwell-Luc operation on left antrum under general ($CHCl_3$) anesthesia; antral mucosa polypoid; severe bleeding throughout operation. Heart failure occurred as last stitch was inserted into mouth wound; abdomen opened and heart massaged but death occurred.

AFFECTIONS OF FAUCES, PHARYNX AND NASOPHARYNX (2015).

Congenital occlusion of choana (right)	1
Foreign bodies in fauces and pharynx (fish bones)	9
Acute tonsillitis	29
Diphtheria	3
Vincent's angina	9
Ulcer (pneumococcal) of tonsil	1
Adenoids and enlarged tonsils	1824
Hypertrophy of lingual tonsil	27
Peritonsillar abscess	22
Acute pharyngitis	8
Ludwig's angina	1
Chronic pharyngitis (including granular pharyngitis)	13
Pharyngitis sicca	7
Keratosis pharyngis	3
Enlargement of uvula, papilloma, bifid uvula	15
Secondary syphilis of fauces and pharynx	4
Tertiary syphilis of fauces and pharynx	6
Lupus of soft palate and pharynx	1
Tuberculous disease of tonsil (proved by microscope)	8
Cyst of tonsil	6
Cyst of nasopharynx	1
Nasopharyngeal fibroma	1
Malignant tumor of tonsil	2
Malignant tumor of fauces and pharynx	5
Malignant tumor of nasopharynx	1
Postdiphtheritic paresis of soft palate	1
Sensory neuroses	7
	2015

AFFECTIONS OF MOUTH (26).

Cleft palate	4
Injury to tongue, fauces or palate	4
Simple ulcer of tongue	1
Simple ulcer of hard palate	1
Submaxillary abscess	1
Alveolar abscess	4
Leucoplakia	2

Tertiary syphilis of tongue, fauces and palate	6
Malignant disease of tongue, alveolus and palate	3
	26

OPERATIONS ON MOUTH, FAUCES AND PHARYNX.

Tonsils and adenoids removed (guillotine and curette) (1 death)	1407
Adenoids only removed	131
Tonsils dissected out (local anesthesia)	143
Tonsils dissected out (chloroform anesthesia)	17
Lingual tonsil removed	1
Peritonsillar abscess opened (excluding those operated on in outpatient department)	17
Foreign body removed from fauces and pharynx	9
Diathermy: (1) to soft palate (including fauces) and tonsil, 4; (2) to tongue and tonsil, 1; (3) to tongue only, 4; (4) to uvula and pharynx, 1	10
Alveolar abscess opened	1
Abscess of hard palate opened	1
	1737

FATAL CASE FOLLOWING REMOVAL OF TONSILS AND ADENOIDS.

J. G., male, aged 24 years. Admitted January 1, 1927, with a history of chronic middle ear suppuration (right) for many years; recently pain over right side of head and giddiness.

Examination.—Right drumhead shows anterior perforation with foul discharge; tonsils enlarged and septic; adenoids present; middle ear deafness on right side. It was decided to remove tonsils and adenoids in first place; operation performed on November 15, 1927, under ethyl chlorid anesthesia with guillotine and curette. Early the following morning the patient suddenly became comatose and died at 8:15 o'clock.

Postmortem.—Temporal lobe abscess on right side (silent area); pus present in both lateral ventricles; basal meningitis present.

AFFECTIONS OF THE LARYNGEAL PHARYNX AND ESOPHAGUS (36).

Traction pouch of esophagus	2
Foreign body in esophagus	10
Abscess of esophagus secondary to foreign body	1
Stricture—	
(1) Simple	4
(2) Malignant	13
(3) Syphilitic	1
Cardiospasm vel achalasia	4
Globus hystericus	1
	36

ESOPHAGOSCOPY.

Examination (one death)	76
Removal of foreign body	10
Dilatation	6
	92

FATAL CASE FOLLOWING ESOPHAGOSCOPY.

J. M., female, aged 65 years. Admitted January 14, 1927, complaining of difficulty in swallowing both solid and liquid food for one year. Indirect examination showed larynx normal; no collection of saliva in pyriform sinuses.

Local anesthesia. Direct esophagoscopy revealed growth in postcricoid region; piece removed and sent for examination. Pathologist stated "appearances suggestive of malignancy but the piece removed was too small to give an opinion." February 17, 1927. Patient very ill; cannot retain rectal salines. February 18, 1927. Death.

Postmortem.—Enlarged gland present on right side of larynx. Examination of esophagus showed margins of wound made at operation to be unhealthy; lungs showed purulent bronchitis and bronchopneumonia; aorta atheromatous; posterior mediastinitis present; gall bladder contained numerous calculi.

Affections of Larynx, Trachea and Bronchi (107).

CONGENITAL AND TRAUMATIC AFFECTIONS OF LARYNX, ETC.

Congenital malformation of larynx	1
Injury to larynx	1
Laryngeal stenosis (one postdiphtheritic, one due to pressure of glands)	2
Foreign body in larynx	1
	5

ACUTE INFLAMMATORY AFFECTIONS.

Acute catarrhal laryngitis	12
Acute edematous laryngitis	1
Acute septic laryngitis (subglottic)	1
Subacute catarrhal laryngitis	6
	20

CHRONIC INFLAMMATORY AFFECTIONS.

Chronic catarrhal laryngitis	13
Laryngitis sicca	8
Bronchiectasis	1
Vocal nodules	1
Pachydermia of larynx	4
Tuberculosis of larynx	16
Lupus of larynx (one case had also lupus of palate)	2
Syphilis (tertiary) of larynx	2
	47

TUMORS.

Simple: Papilloma	3
Fibroma	3
Myxomatous cyst	3
Malignant: Intrinsic	2
Extrinsic	4
	15

NERVOUS AFFECTIONS OF LARYNX.

Functional aphonia	7
Abductor paresis	5

Recurrent paralysis (complete), right	2
Left	4
Bilateral	1
Sensory laryngeal neurosis	1
	20

OPERATIONS ON LARYNX, TRACHEA AND BRONCHI.

Direct laryngoscopy: Examination only	11
Direct laryngoscopy with operation:	
Dilatation of laryngeal stenosis (sittings)	26
Simple tumor removed from vocal cord	7
Diathermy to epiglottis	2
Intratracheal catheter introduced (one death)	2
Intratracheal swab for making vaccine in asthma cases	30
Foreign body removed from larynx	1
Cautery to tuberculous larynx	2
Bronchoscopy	4
Bronchiectasis (sittings)	3
Tracheotomy	7
Thyrotomy	1
Intubation	1
	97

FATAL CASE FOLLOWING INTRODUCTION OF INTRATRACHEAL CATHETER.

A. S., male, aged 46, admitted November 21, 1927. Patient had been treated elsewhere for postcricoid carcinoma by means of deep X-ray therapy. He was traveling from the north of Scotland today when he had a severe hemorrhage from his throat. He has had three recent hemorrhages.

Examination.—Larynx shows a large ulcerated growth in region of right pyriform sinus from which the hemorrhage has evidently come. Mass of malignant glands on left side of neck; pulse 120; morphia and hemoplastin given; ice to suck. January 26, 1927. Patient has had two moderately severe hemorrhages since admission; attempt to apply diathermy unsuccessful owing to excessive hemorrhage. Intratracheal anesthesia tube passed through larynx to give breathing space. No

excessive bleeding following operation. January 29, 1927. Patient developed pneumonia and died today. No postmortem obtained.

Affections of the Ear (2142).

1. THE EXTERNAL EAR.

Congenital malformation	3
Stenosis of external auditory meatus	4
Injury to external ear (one burn)	4
Hematoma of auricle (following injury)	1
Fracture of temporal bone	4
Injury to tympanic membrane	5
Cerumen	532
Foreign body in ear	16
Otitis externa diffusa	121
Furunculosis	91
Perichondritis of auricle (three following radical mastoid operation)	4
Septic cyst of auricle	1
Mastoid gland abscess	6
Erysipelas of auricle	1
Papilloma of external auditory meatus	1
Sebaceous cyst of auricle	1
Exostosis and hyperostosis	5
*Malignant disease of external auditory meatus	1
	801

MALIGNANT DISEASE OF EXTERNAL AUDITORY MEATUS (LEFT) INVOLVING AURICLE, SKIN OVER MASTOID PROCESS AND ALSO MASTOID BONE.

L. I., female, aged 47. First seen June 22, 1927. History: Discharge from left ear for years; cause unknown; occasional slight pain in left ear; deafness not severe; no sickness or giddiness.

Examination.—Absolute stenosis of left external meatus, which bleeds readily; the skin of the meatus and auricle were of almost cartilaginous hardness; pus in meatus; no facial

*Case recorded.

paralysis; slight tenderness over left mastoid; with noise box in right (good ear) patient is not quite deaf. X-rays showed outline of cells on left side hazy as compared with right side. Patient admitted for observation.

June 28, 1927. Radical mastoid operation (left). Skin over mastoid so hard that it cracked as flap was dissected forwards; periosteum of mastoid infiltrated with the growth; mastoid also involved; bone soft; no pus in cells or antrum; dura of middle fossa exposed by disease in roof of cavity; malleus and incus healthy; removed with drumhead; Koerner flap cut out—even then one had not reached healthy tissue; large piece of infiltrated skin removed from skin flap over mastoid; anterior meatal wall very slightly affected; wound packed and left open; tissue sent for examination. Pathologic report: "Tissue shows widespread carcinoma infiltrating the muscle. In areas the neoplasm resembles a basal celled carcinoma while in other areas acinus formation has commenced, resembling an adenocarcinoma."

July 14, 1927. Patient has had five doses of X-rays; wound granulating well; discharged.

March 23, 1928. Letter received from patient's doctor saying that patient died on March 16, 1928, probably from extension of disease into temporal lobe of brain.

II. AFFECTIONS OF MIDDLE EAR CLEFT.

Injury to middle ear	3
Hematotympanum following injury	4
Eustachian obstruction	48
Acute nonsuppurative otitis media	72
Chronic nonsuppurative otitis media	39
Acute suppurative otitis media: Right	69
Left	57
Bilateral	15
Acute suppurative otitis media with mastoid complication—	
Right	25
Left	15
Bilateral	1
Chronic suppurative otitis media: Right	133
Left	131
Bilateral	74

Chronic suppurative otitis media with mastoid complication—	
Right	37
Left	42
Bilateral	1
Sequelae of suppurative otitis media: Right	122
Left	123
Bilateral	87
Simulated mastoiditis	1
Tuberculous otitis media	1
Exostosis of promontory	1
*Malignant disease of middle ear	2
Otalgia of unknown origin	5
	1108

No. 2.—E. L., female, aged 50 years, first seen December 12, 1910. The patient was operated upon in 1910 for malignant disease of left middle and inner ear. At operation a polypus growing from the region of the facial canal was removed. The tumor had eroded the facial canal, and the patient had a peripheral paralysis of the right facial nerve. Radical mastoid operation completed and patient made a good recovery. Pathologic report upon polypus: "Sarcoma probably of endothelial type growing from lymph spaces."

The patient was asked to report on several occasions, but did not come again until sent by her doctor on December 28, 1927—i. e., seventeen years later, complaining of bleeding growth in right ear, pain and giddiness.

Examination showed a hard mass on the right side, below the tip of the mastoid, and also a swelling of the right tonsillar region and lateral wall of the pharynx; tongue protruded to right; loss of taste on right side of tongue; right vocal cord does not move; patient unable to put right hand to back of head.

Neurologic examination (Professor Edwin Bramwell): Ptosis of right eye; right pupil smaller than left (cervical sympathetic involved); optic discs normal; no paresis of ocular muscles; some loss of sensation on right side of face; complete right facial paralysis; right sternomastoid reduced to

*Cases recorded. For No. 1, see under "External Ear."

thin band and trapezius also wasted. No weakness, incoordination or abnormality of the reflexes in either upper or lower limbs. Patient admitted for further investigation.

January 9, 1928. Patient developed acute intestinal obstruction and was transferred to surgical ward for operation. Death after abdominal operation.

Postmortem.—Endothelioma invading base of skull and posterior and middle cranial fossa on right side.

III. Otosclerosis.

Otosclerosis	48
Mixed middle and inner ear deafness (probably advanced otosclerosis)	25
	73

IV. Labyrinthine and Intracranial Complications of Suppurative Otitis Media.

Six cases complicating acute suppurative otitis media:

Labyrinthitis	1
Perisinus abscess	4
Perisinus abscess and sinus thrombosis	1
	6

All recovered.

CASE RECORDS.

(The four cases of perisinus abscess are not recorded in detail.)

Case 1. Foreign Body in Right Ear; Injury to Tympanic Membrane; Acute Suppurative Otitis Media; Labyrinthitis; Recovery.—M. H., female, aged 7 years. Admitted September 23, 1927, with a history of having put a bead into her right ear two days ago. Child's doctor reports that an attempt had been made to remove the bead. Child removed to local hospital, where a further unsuccessful attempt was made to remove the bead. When taken home afterwards the child was very sick but her mother did not notice any giddiness.

Examination, September 23, 1927. Right external meatus contains blood. Anesthetic given and bead removed; child kept in bed.

September 24, 1927. Child very sick last night; giddiness on sitting up; nystagmus to left (healthy) side; with noise box going in left (good) ear child is quite deaf; no neck rigidity; Kernig positive; no facial paralysis.

September 25, 1927. No fever; nystagmus to left still present. Child slowly improved, and on October 14, 1927, was able for the first time to hear a loud voice by the right ear, with the noise box going in the left ear.

October 26, 1927. Sent to Convalescent House.

November 11, 1927. With finger in left ear patient hears conversation voice at one foot; cold caloric test with the Dundas-Grant apparatus produced nystagmus to left in 40 seconds.

May 26, 1928. With noise box going in left ear child can hear raised voice but cannot distinguish words.

Case 2. Acute Suppurative Otitis Media (Right) with Mastoid Complication; Perisinus Abscess; Sinus Thrombosis; Recovery.—M. M., female, aged 13 years. Admitted October 19, 1927, with history of discharge from right ear for three weeks; pain commenced in right ear three days ago and kept patient awake last night; vomiting present.

Examintaion: Sagging of posterior meatal wall on right side; drumhead not seen; hearing moderate in right ear.

October 20, 1927. Schwartze operation (right ear): Fistula through mastoid cortex; lateral sinus exposed, covered with granulations; upper part of wound closed.

October 23, 1927. Temperature swinging; patient has had several rigors.

Second Operation.—Sinus exposed for one and a half inches behind the upper knee; gauze packed between sinus and bone and sinus slit up; mural clot present; wound left open.

October 25, 1927. Temperature subsiding; no rigors; wound dressed. Opening in sinus had healed and circulation had become reestablished; sinus slit up further and lumen of sinus packed; no pus seen.

November 22, 1927. Patient made a satisfactory recovery and went to Convalescent House today.

Twenty-four cases complicating chronic suppurative otitis media:

Circumscribed labyrinthitis	3
Serous	2
Induced	1
Acute diffuse purulent labyrinthitis	2
Latent labyrinthitis	1
Spontaneous cure of labyrinthitis	3
Extradural abscess in middle fossa	2
Extradural abscess in middle fossa and circumscribed labyrinthitis	1
Perisinus abscess	1
Sinus thrombosis	1
Perisinus abscess and sinus thrombosis	1
Perisinus abscess and cerebellar abscess	1
Perisinus abscess, sinus thrombosis, latent labyrinthitis and cerebellar abscess	1
Perisinus abscess, sinus thrombosis, cerebellar abscess and meningitis	1
Perisinus abscess, septicemia, temporal lobe abscess and purulent meningitis	1
Perisinus abscess, acute diffuse purulent labyrinthitis and purulent meningitis	1
Acute purulent labyrinthitis and purulent meningitis	1
	24

Seventeen recovered. Seven died.

Recoveries:

Circumscribed labyrinthitis	3
Serous labyrinthitis	2
Induced labyrinthitis	1
Acute diffuse purulent labyrinthitis	1
Latent labyrinthitis	1
Spontaneous cure of labyrinthitis	3
Extradural abscess in middle fossa	2
Extradural abscess in middle fossa and circumscribed labyrinthitis	1
Perisinus abscess	1
Sinus thrombosis	1
Perisinus abscess and cerebellar abscess	1
	17

Deaths:

Perisinus abscess and sinus thrombosis	1
Perisinus abscess, sinus thrombosis, cerebellar abscess and meningitis	1
Perisinus abscess, septicemia, temporal lobe abscess and meningitis	1
Acute purulent labyrinthitis and purulent meningitis	1
Purulent meningitis	1
Perisinus abscess, sinus thrombosis, latent labyrinthitis and cerebellar abscess	1
Perisinus abscess, acute purulent labyrinthitis, purulent meningitis	1
	7

RECORDS OF THE CASES WHICH RECOVERED.

Note.—The following twelve cases have not been recorded in detail: Three cases of circumscribed labyrinthitis; two cases of serous labyrinthitis; three cases of spontaneous cure of labyrinthitis; two cases of extradural abscess in middle fossa; one case of extradural abscess in middle fossa and circumscribed labyrinthitis; one case of perisinus abscess.

Case 1. Chronic Suppurative Otitis Media (Left); Radical Mastoid Operation (L.); "Induced" Labyrinthitis; Labyrinth Operation; Recovery.—G. Y., male, aged 7 years. Seen November 20, 1927, with history of pain in and discharge from the left ear of two years' duration; severe pain for five days with rigor and sweating but no vomiting.

Examination: Temperature on admission 102° F.; slight tenderness at mastoid tip; granulations and cholesteatoma in meatus; whisper heard by left ear at six inches; no spontaneous nystagmus; no rigidity of neck.

November 24, 1927.—Radical mastoid operation left ear; pus in antrum; lateral canal prominence normal; cavity skin grafted.

November 25, 1927. Temperature 101° F.; patient has had a bad night; is very sick and has marked nystagmus to the right; complete deafness in left ear, with noise box going in right ear.

Second Operation.—Skin graft removed; promontory chipped off and lateral canal opened (Hinsberg operation); wound left open.

November 30, 1927. Child much better; nystagmus only of first degree; wound closed.

December 20, 1927. Patient sent to Convalescent House.

February 6, 1928. Operation cavity satisfactory.

Case 2. Chronic Suppurative Otitis Media (Left); Acute Purulent Labyrinthitis; Radical Mastoid and Hinsberg Labyrinth Operation (L.); Recovery.—D. M., male, aged 33 years. First seen in May, 1926, when a polypus was removed from the left ear after the inner ear had been tested. Patient returned March 6, 1927, suffering from attacks of giddiness, during which he could not stand upright and felt as if he were moving to the left; intermittent earache and tinnitus in left ear; no headache.

Examination showed a polypus and foul pus in left meatus; Weber not lateralized; Rinne absolutely negative on left side; with noise box going in right ear he is quite deaf. Well-marked spontaneous horizontal and rotatory nystagmus to the right (sound) side; no spontaneous pointing error; no fistula symptom; on Romberg's test he tends to fall to left; no dysdiadokokinesia; patient can recognize and name common objects. Examination of central nervous system negative.

March 8, 1927. Patient has vomited twice since admission. Radical mastoid and labyrinth operation left ear; cholesteatoma in antrum, aditus and attic; fistula in lateral canal with dark purple contents; canal opened and promontory removed with gouge; wound left open. Patient made good progress, and on March 11, 1927, the wound was closed with stitches.

March 24, 1927. No fever or nystagmus.

April 5, 1927. Patient sent to Convalescent Home.

Case 3. Chronic Suppurative Otitis Media (Right); Latent Labyrinthitis; Radical Mastoid and Hinsberg Labyrinth Operation; Recovery.—A. H., male, aged 27 years. First seen May 9, 1927, with history of discharge from right ear of many years' duration, following measles, and increasing deafness; attacks of giddiness, during which external objects rotate.

Examination showed foul pus and polypus in right external meatus; Rinne negative right side; with noise box going in

left (good) ear patient quite deaf; no spontaneous nystagmus; slight swaying on Romberg's test; cold syringing right ear gave no reaction; no fever.

May 10, 1927. Radical mastoid and Hinsberg labyrinth operation right ear; pus and granulations in antrum; lateral canal prominence eroded; granulations sprouting out; promontory removed; granulations in vestibule; wound left open.

May 13, 1927. No fever; spontaneous nystagmus to both sides; no vertigo; wound closed with stitches.

June 8, 1927. The nystagmus has gradually disappeared; patient discharged.

Case 4. Recurrent Acute Suppurative Otitis Media (R.); Schwartze Operation (R.); Sinus Thrombosis; Recovery.—R. W., male, aged 20 years, had the following history: Tonsils and adenoids were removed in 1921. At that time the right drumhead showed a scar posteriorly. The patient returned on April 18, 1927, complaining of pain in and discharge from the right ear. Examination showed pus in the right external meatus; posterior perforation of right drumhead; no mastoid tenderness; conservative treatment advised.

April 27, 1927. Patient admitted with severe pain in right ear; no neck rigidity; no spontaneous nystagmus; with finger closure of left ear he hears raised voice at six inches only; temperature 99° F.; pulse 100. Schwartze operation; pus under pressure in antrum; diseased cells over lateral sinus; sinus and middle fossa dura exposed with gouge and found healthy.

April 28, 1927, to May 3, 1927. Temperature very suggestive of sinus thrombosis; veins of both optic discs full and tortuous; blood culture negative.

Second Operation.—Wound opened up and cross cut made backwards towards torcular end of sinus; granulations on sinus; bone removed in backward direction till healthy sinus reached; gauze packing inserted between sinus groove and vessel wall; sinus slit; free bleeding; mural thrombus; bulbar end packed and cavity of sinus laid open and packed; wound left open.

May 5, 1927. Temperature (evening) 105° F.; blood culture showed a hemolytic streptococcus; lumbar puncture—cerebrospinal fluid clear but under pressure; white blood corpuscles

16,250 per cm.; 50 cc. of 10 per cent sodium chlorid given intravenously.

June 6, 1927, to June 17, 1927. Rapid improveemnt; sent to Convalescent House.

January 23, 1928. Patient reports dry ear; with finger closure of left ear he can hear whisper by right ear at two feet.

Case 5. Chronic Suppurative Otitis Media (Bilateral); Perisinus Abscess (Left); Radical Mastoid Operation (L.); Cerebellar Abscess; Recovery.—F. M., male, aged 15 years, sent in April 13, 1927, with the following history from his doctor: "Three weeks ago he was seized with headaches, vomiting, high temperature and was sent to the local hospital as a possible cerebral abscess. He had had otorrhea for years but the discharge had suddenly stopped. He was sent back from hospital as 'not a mastoid' and has been under observation since. The main symptoms yesterday were very severe occipital headache and vomiting. For three weeks there has been giddiness and a tendency to fall over; there is nystagmus to both sides."

Examination shows granulations in the right drumhead growing through a posterior marginal perforation. The left meatus contains very foul pus; posterosuperior marginal perforation of left drumhead; Weber not lateralized; Rinne positive (right), negative (left); with noise box going in right ear patient is not quite deaf in the left ear. Coarse nystagmus to left; fine nystagmus to right; pupils dilated and equal; tongue dry and furred; sordes on teeth; patient is sleepy and has to be roused to answer questions; no pain on tapping over skull; no inability to recognize and name objects; knee jerks present and active; Babinski—flexor response; no Kernig; abdominal reflexes absent on left side; urine normal; no pathologic change in optic discs.

Operation.—Radical mastoid operation (left); cholesteatoma in antrum; extradural perisinus abscess found, the posterior wall of the abscess cavity being formed by the lateral sinus; wound left open.

April 15, 1927. Wound dressed, looking healthy; both ocular fundi normal; lumbar puncture—cerebrospinal fluid clear but under pressure; cells scanty; no growth obtained on culture.

April 19, 1927. Severe pain in left side and back of head;

vomited; cannot locate tip of nose with left forefinger when eyes are closed; extreme loss of power on left side; abdominal reflexes absent; early optic neuritis (L.); nystagmus both to right and left but none in middle line.

Second Operation.—Posterior vertical and lateral canals opened up and vestibule opened; dura of posterior fossa slit medial to sinus but no pus escaped; forceps passed into cerebellum and large cerebellar abscess evacuated; abscess drained with iodoform gauze.

April 23, 1927. Pointing test on left side shows marked incoordination; grasp of left hand weaker; bilateral papilledema and retinal hemorrhage on right side.

April 24, 1927. Patient sleepy; vomiting has recurred.

Third Operation.—Incision made downwards and backwards to expose occipital fossa; bone removed; cerebellar dura incised horizontally and abscess opened from behind so that thorough drainage was obtained.

April 4, 1927, to May 2, 1927. Very gradual improvement.

May 19, 1927. Patient got up; no spontaneous falling.

May 24, 1927. Patient not so well; vomiting and giddiness; nystagmus to left; dysdiadokokinesia; almost complete loss of power of left hand; marked incoordination of left side.

Fourth Operation.—Granulations scraped away; cerebellum bulges into wound; cerebellum opened with sinus forceps and some sloughy brain tissue came away. Following this, progress slow but satisfactory.

May 31, 1927. Fifth Operation.—Wound opened up but drain into cerebellar abscess not reinserted, as there had been no vomiting for two days. Granulations removed from right ear with ring knife.

July 1, 1927. Patient sent to Convalescent House under strict observation.

January 18, 1928. Patient reported; no complaint; to begin work again.

CASE RECORDS OF SEVEN DEATHS.

Case 1. Chronic Suppurative Otitis Media (Left); Perisinus Abscess; Sinus Thrombosis.—D. B., male, aged 15 years. Admitted October 10, 1927, with history of discharge from left ear for several years, following measles; pain in and swelling behind left ear, with headache, fever and giddiness for

one week. Patient has been in bed for seven weeks; no definite rigors.

Examination showed pus, granulations and cholesteatoma in left external meatus; fluctuating swelling over left mastoid. Right ear normal. With noise box in healthy ear patient is not deaf; no spontaneous nystagmus; after two minutes' cold syringing of left ear, fine vertical nystagmus produced and patient falls backwards; no rigidity of neck; no Kernig; no pointing error; no dysdiadokokinesia; no falling of uplifted hands; grasp of hands strong and equal; tongue furred; temperature 99° F., pulse 104. Dr. Fraser reports "Patient does not look flushed, frightened and restless like a case of meningitis, nor is he very drowsy; indeed one cannot make a lightning diagnosis of the intracranial complications present."

Radical Mastoid Operation (L.) on Day of Admission.—Mastoid excavated by cholesteatoma; granulations on sinus; large extradural perisinus abscess evacuated after making an incision backwards; sinus slit, pus in lumen, coming up from bulb region; sinus laid open; no evident clot; wound left open. Lumbar puncture—fluid clear, under pressure; cells 13 per cm.; 85 per cent polymorphs.; chlorids (as NaCl); 661 mgs. per cent; protein 57 mgs per cent; sugar 30 mgs. per cent.

October 12, 1927. Temperature swinging.

Second Operation.—Left internal jugular vein ligated; sinus further exposed in backward direction but end of septic clot not reached.

October 13, 1927, to October 17, 1927. Hectic temperature; progressive drowsiness; internal jugular vein washed out.

October 20, 1927. Third Operation.—Cerebellum explored for abscess; no pus found.

October 26, 1927. Death; postmortem refused.

Case 2. Chronic Suppurative Otitis Media (Left); Cerebellar Abscess; Meningitis; Lateral Sinus Obliterated.—A. M., male, aged 16 years. Admitted to City Fever Hospital on August 13, 1927, with diagnosis of diphtheria—proven negative; discharge from left ear; pain behind left ear and in back; head retraction; neck rigidity; Kernig positive; lumbar puncture—cerebrospinal fluid under tension contained many polymorphs. Patient improved with repeated lumbar punctures and on September 12, 1927, the cerebrospinal fluid was clear but

still under tension; blood culture negative; Kernig positive: head retraction and Kernig still present but less marked.

September 13, 1927. Patient admitted to Royal Infirmary. On examination patient very noisy; severe headache; marked head retraction; Kernig positive; temperature subnormal; pulse 50; left meatus full of pus; no trace of drumhead left; some pain on pressure over left antrum. Radical mastoid operation left ear; large cerebellar abscess evacuated. Lumbar puncture —cerebrospinal fluid clear but under pressure.

September 15, 1927. Patient alternately noisy and comatose; abscess draining well. September 19, 1927. Death.

Postmortem.—Cerebellar abscess; purulent meningitis; obliteration of left lateral sinus, probably by an old infection.

Case 3. Chronic suppurative otitis media (right); cholesteatoma; Perisinus Abscess; Temporal Lobe Abscess; Meningitis.—J. G., male, aged 31 years. First seen October 11, 1913, with deafness in and discharge from the right ear for twelve months; middle ear suppuration (right) found and conservative treatment advised; tonsils and adenoids removed.

February 23, 1927. Patient admitted in moribund condition; relatives say he has had pain in right ear for some months; headaches latterly and for the last week restlessness, irritability and sleeplessness; no vomiting or rigors.

Examination: Temperature 103.8°; pulse 88; patient semicomatose; answers are not reasonable; pain over right side of head and also on tapping over right temporal lobe and cerebellar fossa; right pupil dilated; patient is right handed and could name simple objects (e. g., key, scissors) but refused to name other objects; Kernig positive on both sides; marked neck rigidity; patient moves both arms but moves the right more freely than the left; patient too ill for any further tests. Lumbar puncture—fluid blood stained, not under tension. Operation (same day)—Incision for radical mastoid operation (right); cholesteatoma, granulations and pus in antrum; middle fossa dura exposed with gouge—thickened and tense; no extradural abscess; perisinus abscess was exposed with granulations on sinus; dura of posterior fossa therefore separated in inward direction up to posterior semicircular canal and opened —no abscess found; dura of middle fossa then opened and

large temporal lobe abscess evacuated; drain inserted and wound left open.

Progress following operation satisfactory till March 2, 1927, when temperature rose to 102° F., with rigor; double optic neuritis; lumbar puncture unsuccessful.

March 5, 1927. Has had typical rigors and sweating; no vomiting; no retention of pus in brain abscess.

Second Operation.—Sigmoid sinus opened; wall healthy; no thrombosis; blood culture negative.

March 9, 1927. Temperature normal since second operation.

March 26, 1927. Has had continual rigors and swinging temperature since March 10, 1927 (blood infection?). Blood culture—Gram negative bacillus present; 2.5 gm. electrargol given intravenously. Patient went slowly downhill and died on April 18, 1927, after being comatose for several days.

Postmortem.—Temporal lobe abscess; purulent meningitis (widespread); some old loose clot in right lateral sinus.

Case 4. C. O. M. S.; Radical Mastoid Operation; Labyrinthitis; Meningitis.—T. W., male, aged 21 years. Previously seen in 1911 and 1916, with suppurative otitis media in the left and right ears, respectively, which cleared up in each case with conservative treatment. Patient returned on April 22, 1927, when the right ear was found to be dry; the left ear contained pus and a polypus. Inner ear tested and polypus removed from left ear; left ear treated by zinc ionization. September 21, 1927. Pain; purulent discharge in right ear with recurrence of polypus.

September 22, 1927. Radical mastoid operation left ear; skin graft applied.

September 23, 1927. Frequent vomiting; nystagmus to right (sound) side; with noise box in right ear patient is quite deaf; no rotation of external objects; temperature 97.6° F., a. m., 102° F. p. m.

September 24, 1927. Wound suppurating (patient has a suppurating sore on his left hand); stitches removed; wound opened up; packing and graft removed; eusol gauze dressing.

September 27, 1927. Left ear still quite deaf with noise box going in right ear; giddiness on sitting up; nystagmus to right; no neck rigidity; no Kernig.

Second Operation.—Hinsberg labyrinth operation and translabyrinthine drainage; promontory chipped off and vestibule opened; stapes healthy; pus in vestibule; lateral canal opened. Lumbar puncture then performed; fluid under slight tension and not clear. Internal meatus opened with gouge; free flow of cerebrospinal fluid obtained; wire drain inserted. Pathologist's report on cerebrospinal fluid—360 cells per cm.; cells mainly polymorphs (91 per cent); no growth obtained.

September 29, 1927. Temperature 103° F.; severe headache. Lumbar puncture—Cerebrospinal fluid contains streptococci and pus.

October 1, 1927. Death.

Postmortem.—Purulent meningitis (streptococcal), following otitis media; no thrombosis of lateral sinus.

Case 5. C. O. M. S.; Radical Mastoid Operation; Meningitis.—R. C., male, aged 12 years. Admitted February 18, 1927, with foul smelling discharge from left ear for two years following measles; recent pain and deafness in left ear and fever.

Examination: Thick discharge and granulations in left meatus; very little remains of left drumhead; swelling over tip of left mastoid, with extreme tenderness; middle ear deafness on left side.

February 19, 1927. Radical mastoid operation left ear; middle fossa dura and sinus exposed with gouge, healthy; cavity skin grafted.

February 21, 1927. Temperature 100° F.; complains of giddiness.

February 22, 1927. Giddiness worse; nystagmus to right; temperature 102.4° F. Lumbar puncture—fluid turbid and under pressure (cells 750 per cm., mostly polymorphs; on culture growth of staphylococcus albus and streptococcus obtained; moderate increase of globulin.)

Second Operation.—Hinsberg labyrinth operation and translabyrinthine drainage; free flow of cerebrospinal fluid obtained. (Culture from labyrinth showed a pure growth of streptococci.)

February 23, 1927, and February 24, 1927. Daily lumbar puncture; hemolytic streptococcus found on direct films of cerebrospinal fluid and also on culture; cells very numerous,

mainly polymorphs; sugar 77 mgs. per cent; chlorids 655 mgs. per cent; phosphates 3.94 mgs. per cent. Death February 25, 1927. No postmortem obtained.

Case 6. C. O. M. S.; Labyrinthitis; Perisinus Abscess; Sinus Thrombosis; Cerebellar Abscess (Double Abscess).—D. E., male, aged 13½ years. Admitted October 24, 1927, with a history of intermittent discharge from both ears for five years; right ear became very painful a week ago, when the discharge stopped; for one week he has been kept in bed with severe headache, high swinging temperature, vomiting and rigors; also pain over right mastoid and attacks of giddiness recently.

Examination: Right ear contained pus; large central perforation of right drumhead; mastoid tenderness at tip of right mastoid. Left external meatus contains foul pus, granulations and cholesteatoma; small piece of lead (from a pencil) lying up against the inner wall of left tympanum (the drumhead has disappeared); Schwabach lengthened; Weber not lateralized; Rinne negative both sides; with noise box going in left ear the right ear is quite deaf; with noise box going in the right ear he hears conversation voice at two feet by the left ear. No spontaneous nystagmus; no Rombergism; no Kernig; no stiffness of neck; pupils equal and react equally and well to light and accommodation; no astereognosis; no sensory aphasia (patient is right handed); no dysdiadokokinesia; no weakness of grasp on either side; no incoordination of upper and lower limbs; superficial and deep reflexes equal and active; heart, lungs and abdomen negative; urine normal; temperature 98.8° F.; pulse 88; respirations 20; W. B. C., 22,000 per cm.; tongue furred. Both ears prepared for operation on same day.

Modified radical mastoid operation left ear. After removing small piece of lead; antrum healthy; patient's general condition evidently not due to left ear; wound closed.

Radical mastoid operation right ear; pus and cholesteatoma in antrum; large perisinus abscess; lateral canal opened up by cholesteatoma; Hinsberg labyrinth operation performed; lateral sinus then further exposed by cross cut backwards and bone removed until healthy bluish sinus wall was exposed; granulations on anterior wall of sinus; sinus slit up after packing off both ends and clot turned out; anterior wall of sinus

exposed and cavity packed; internal jugular vein not ligatured; wound left open.

October 25, 1927. No fever; some sickness.

October 26, 1927. Evening temperature 103° F.; sickness continues; lumbar puncture—fluid clear and not under pressure (7 cells per cm.; chlorids 667 mgs. per cent; sugar 84 mgs. per cent).

October 27, 1927. First dressing; left ear looking well, cavity repacked; right ear—packing removed from bulbar end of sinus; no bleeding but pus coming up between sinus wall and bone; packing removed from cavity of sinus, posterior wall healthy; packing then removed from torcular end; free gush of blood so fresh packing reinserted between sinus wall and bone. Bezold's abscess at tip of mastoid opened up.

October 28, 1927, to November 7, 1927. Temperature dropped and is now subnormal; no rigor; pus continues to well up from region of jugular bulb.

Second Operation.—Right internal jugular vein ligated in the neck; no attempt made to wash through the vein.

November 8, 1927, to December 2, 1927. Progress fairly satisfactory but has often severe headache; no fever; occasionally vomits; losing weight; bilateral papilledema; no tubercles seen on choroid; knee jerks absent; Babinski—extensor response on left side. Lumbar puncture—fluid clear, not under tension (cells 20 per cm.; no organisms seen on direct smears; no growth in culture).

December 3, 1927, to December 12, 1927. Has vomited frequently; very drowsy; now almost comatose; temperature subnormal; respirations 10 to 16 per minute; pulse slow (56); headache severe; both pupils dilated; all objects which are shown to him patient calls "pens." Babinski—extensor response on left (contralateral) side; seen by neurologist, who diagnosed temporal lobe abscess.

Third Operation.—Dura of temporal lobe exposed; rather tense and bulging but otherwise quite normal. It did not look as if there was an abscess behind it. Dura incised and temporal lobe explored—no abscess found; brain prolapsed; incision then made on inner wall of sinus and dressing forceps introduced, and a large cerebellar abscess containing foul pus

under great tension exacuated and drained. Following this operation patient did well for a time but later went gradually downhill and died on January 19, 1928, after being unconscious for three days.

Postmortem.—On section of cerebellum two large abscess cavities were found in the right lobe; these were each about the size of a walnut and were completely separated from each other by a narrow septum. The abscesses contained a considerable quantity of thick, greenish yellow pus. The remainder of the cerebral tissue showed no marked abnormality.

Case 7. C. O. M. S.; Labyrinthitis; Meningitis.—W. H., made, aged 14 years. Admitted December 20, 1927, with history of discharge from right ear for some years; lately severe pain in right ear.

Examination: Erection of right auricle; edema of skin and subcutaneous tissues over right mastoid; intense mastoid tenderness; right meatus contains polypus, mucopus and cholesteatoma; ossicles of left ear well seen through a dry perforation. With noise box going in left ear patient hears conversation voice by right ear at six inches; no spontaneous nystagmus; no facial paralysis; temperature 99° F.

Radical mastoid operation right ear. First blow of chisel liberated thick, curdy pus, which pulsated out under great tension; sinus widely exposed by cholesteatoma and covered with thick, healthy granulations; sinus not opened; no skin graft; wound left open.

December 24, 1927. No fever; Koerner flap cut and wound closed.

December 28, 1927. Headache; giddiness; nystagmus to left; well marked stiffness of neck. With noise box going in left ear patient is quite deaf; temperature 102.8° F. Lumbar puncture—Cerebrospinal fluid turbid and under tension (2600 cells per cm., mainly polymorphs; globulin moderately increased; no organisms present). In spite of this, it was decided to perform translabyrinthine drainage.

Second Operation.—Hinsberg labyrinth operation and translabyrinthine drainage; wound opened up and cleaned out; well-marked fistula in lateral canal, which was further opened up; promontory chipped off and opening made through inner wall of vestibule into internal meatus, where pus was present; only

a feeble flow of cerebrospinal fluid obtained; wire drain inserted.

December 29, 1927. Patient seems wonderfully well except for headache. Lumbar puncture—fluid turbid but under less tension; wound dressed; no flow of cerebrospinal fluid obtained.

December 31, 1927. Wound dressed; profuse flow of cerebrospinal fluid from internal meatus. Lumbar puncture—cerebrospinal fluid turbid and under tension (1100 cells per cm.; globulin increased; direct smear showed Gram positive cocci in chains; a coarse hemolytic streptococcus was obtained on culture).

January 1, 2 and 3. Patient very noisy; no drainage from wound until internal meatus was opened up with labyrinth spoon; hectic temperature; periods of unconsciousness.

January 4, 1928. Death.

Postmortem.—Widespread purulent meningitis.

AFFECTIONS OF THE INTERNAL EAR (138).

Congenital deafness (including deafmutism)	14
Traumatic deafness	2
Bullet injury to inner ear	1
Occupational deafness	2
Senile nerve deafness	9
Labyrinthitis (see "Labyrinthine and Intracranial Complications")	
Congenital syphilitic deafness (one unilateral)	4
Acquired syphilitic deafness	6
Nerve deafness (including cases due to toxemia)	47
Ménière's syndrome (including two syphilitic cases)	7
Tumor of eighth nerve	2
Tinnitus	43
Hysterical deafness	1
	138

OPERATIONS ON THE EAR.

Gland abscess over mastoid opened	5
Furuncle opened	31
Foreign body removed	16
Paracentesis	58
Granulations curetted	10

Polypi removed	46
Plastic	5
Schwartze operation	44
Modified radical mastoid operation	14
Radical mastoid operation	85
Hinsberg labyrinth operation	10
Neumann's labyrinth operation	3
Translabyrinthine drainage	4
Extradural abscess (middle fossa)	2
Extradural perisinus abscess	13
Sigmoid sinus opened	5
Internal jugular vein ligated	2
Temporal lobe explored (negative)	2
Temporal lobe abscess	1
Cerebellum explored (negative)	2
Cerebellar abscess	3
	361

The following two cases have been included in order to complete the list of deaths in 1927. The deaths were, however, not due to disease of the ear.

Case 1. Meningitis of Unknown Origin.—E. B., female, aged 12 years, admitted in unconscious condition on December 26, 1927. History of pain in right ear; frontal headache and vomiting for one week; no otorrhea.

Examination: Sordes on tongue; septic teeth; drumheads indrawn; no mastoid swelling; pupils equal and react to light; temperature 101° F.; pulse 144; respirations 38. Patient died four hours after admission.

Postmortem.—Meningitis (pneumococcal) over vertex; no brain abscess; no primary focus of infection found in ear or elsewhere.

Case 2. Cerebrospinal Meningitis.—A. B., male, aged 23 years. First seen June 15, 1923, when left radical mastoid operation was performed, following chronic suppurative otitis media (left); skin graft applied; recovery.

May 11, 1927. Patient sent back with headache and vomiting of two days' duration.

Examination: Patient noisy and restless; neck rigidity present; temperature 101° F.; pulse 76. Lumbar puncture—Cerebrospinal fluid purulent and under great tension. Without waiting for bacteriologic report the scar behind the left ear was opened up and vestibulotomy and translabyrinthine drainage carried out; profuse flow of cloudy cerebrospinal fluid obtained.

May 12, 1927. Bacteriologic report on blood and cerebrospinal fluid shows Weichselbaum's meningococcus present.

May 13, 1927. Death.

MISCELLANEOUS CASES.

Including dental sepsis, skin diseases, enlarged cervical glands, general surgical cases, negative examinations, etc.	257

ANESTHETICS.

Local anesthesia	673
Nitrous oxid and oxygen	1
Ethyl chlorid	1628
Chloroform and ether	296
Chloroform	25
	2623

Total new patients attending department	4184
Total affections	6669
Total deaths from all causes, including two deaths in 1928	13

II.

THE TREATMENT OF CANCER OF THE PHARYNX, LARYNX AND ESOPHAGUS BY SURGICAL DIATHERMY.*

By Dan Mackenzie, M. D.,

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I do not propose to spend any time on the methods and actions of surgical diathermy, but, assuming with some confidence that you are already familiar with these preliminaries, I will proceed without more ado to discuss its application to cancer of the pharynx, etc.

Doyen of Paris was first to employ diathermy as a destroying agent in cancer; but, so far as our own regions are concerned, it was not until Nagelschmidt introduced it into England that surgical diathermy was seized upon by D. Harmer and others as a means of operating on cancer superior to any other. At all events, it was and is still, deemed to be superior to the ordinary knife, chiefly because of its action in killing cancer cells. In addition, experience has shown that as a bactericide in the tissues of the body, diathermy is also of great service, a discovery that came as a welcome surprise to pioneers.

In operating on pharyngeal cancer, we use diathermy, then, because

1. It prevents the operator squeezing live cancer cells into the lymphatic vessels draining the affected area;

2. It prevents the sowing of the operation field with living cancer cells.

3. It destroys the bacteria of sepsis in the tissues adjoining the field of operation.

I may premise my remarks by noting that diathermy comes into play even in our diagnostic measures. Thus, when we remove a piece of a doubtful growth for microscopic examination, the wound we make should at once be sealed with dia-

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thermy, in order to prevent contamination of the parts around with cancer cells, and secondly, in order to prevent septic infection of the growth itself.

Cancer may start almost anywhere in the pharynx, and although I am unaware of any statistics showing its favorite site of origin, all will agree, I think, that it most frequently makes its first appearanec in or about the tonsil. In my own cases, at all events, I find that the tonsil was affected as often as all the other sites put together. The junction of the anterior faucial pillar with the tongue is also cited as a common starting point, an opinion supported by the frequency with which we find the disease involving tonsil and tongue equally. I have not myself had an opportunity, however, of seeing it actually beginning at this spot. The base of the tongue and the surface of the soft palate seem to furnish sites of origin with about equal frequency, and the hyoid fossa is by no means uncommonly the primary seat. On the uvula and on the posterior wall of the oropharynx it is rare. But further down in the laryngopharynx we encounter postcricoid cancer with apparently increasing frequency. It has the peculiarity of occurring in women oftener than in men, unlike carcinoma of the esophagus proper. Of the two varieties of laryngeal cancer, intrinsic and extrinsic, the former is said to be the more frequent.

From the point of view of the operator we may classify our cases in two varieties, the eradicable and the ineradicable. In the former, the surgeon sets out with the hope and intention of removing every trace of the disease; in the latter, no such expectation can be entertained.

In thus classifying our cases for operation, the situation of the growth must be taken into consideration as well as its type and extent. Early cancer of the tonsil, for example, or of the faucial pillar, or soft palate, or uvula, or intrinsic cancer of the larynx, is more often eradicable than cancer of the base of the tongue, the hyoid fossa, the postcricoid region, or than extrinsic laryngeal cancer.

I propose to take these several situations in their order, and to show in what manner an operator armed with diathermy can deal with the disease.

Before entering into detail, however, we may glance at the principles guiding us in our treatment. The old rule that when

a growth is circumscribed, accessible, and nonadherent, it should be excised in mass, still holds good, at least in my opinion. There are, I know, many operators who content themselves with merely coagulating by diathermy the disease area and removing it piecemeal with the curette. But when we can remove a cancer by an excision that passes entirely through healthy tissue, surely it is safer to do so, as direct coagulation and curetting do not enable us to be quite certain of having destroyed and removed the whole of the growth.

When, however, the disease has infiltrated the tissues so deep as to become adherent, it may be to underlying bone, as when it rests upon the mandible, the hard palate, or the vertebral bodies, and when we are, consequently, unable to undercut it through healthy tissues; or again, when the base of the tongue is deeply involved; or, again, when the cervical glands have become extensively infected or are adherent to each other and to the surrounding structures; then we recognize the cancer to be ineradicable, and all we can do locally is to destroy as much of the disease as possible by diathermy coagulation.

So much for the local growth. We turn now for a moment to consider what should be our action with regard to the cervical gland-bearing region in a case of removable pharyngeal cancer. In this matter there is at the present moment a considerable difference in practice. Some authorities, like Wyeth of New York, see no reason for removing glands and lymphatics unless their hardness to palpation proclaims that they are obviously the seat of secondary cancer. Wyeth points out, what is perfectly correct, that the cervical glands in these cases may be enlarged from simple sepsis only, and he argues that their removal, when they are soft, is therefore unnecessary.

On the other hand, others advocate and practice the removal of the cervical glands and gland-bearing fascia in all cases, even when the glands show no increase in size.

Which of these plans is the more worthy of our support?

In deciding this question, you will perhaps allow me to remind you of the work of Sampson Handley of London in connection with cancer of the mamma. Handley showed that mammary cancer spreads largely, though not of course exclusively, by what he calls "permeation" through the capillary lymphatic vessels. He showed, moreover, that what appear to

the naked eye to be perfectly normal glands frequently contain groups of living cancer cells. That being so, he urged that the operation for the removal of mammary cancer should include not only the visible growth in the mamma, but also, as a routine, the axillary lymphatic glands, and, as far as possible, the lymphatic vessel-bearing tissues that intervene between the primary growth and its most usual sites of metastasis.

Nowadays this sweeping removal, generally known as "block dissection," is the orthodox method of operating, and its advantages are manifest in the improvement in results that has followed its adoption.

Now, have we any reason to suppose that cancer of the pharynx differs from cancer of the mamma in its method of dissemination? If not, then in reverting to the older plan of ignoring possible secondary infections of apparently normal glands, we are obviously falling short of our duty of attempted eradication. In a word, the logical conclusion is that if we aim at a total eradication of the disease, we must remove or destroy not only the primary growth in the pharynx, but, as far as possible, the whole lymphatic area draining the affected pharyngeal locus.

It is said that the removal of uninfected glands removes a "barrier" to the dissemination of a cancerous growth. But is this a fact? Do lymphatic glands present an efficient barrier? Do they not rather provide a nursery for cancer cells? Pathologists say, as a matter of fact, that if lymphatic glands act as a barrier at all, they do so only for a short period, while it is highly probable that they act as a nursery; that in lymphatic glands, in other words, the cancer cells find a nidus that favors their growth and development. In these arguments, however, we must avoid being led astray by metaphors. To call a lymphatic gland either a "barrier" or a "nursery" is beside the point. For, since the experience of cancer operators in other parts of the body shows that the removal of the glands and gland-bearing connective tissue, when it can be accomplished, adds to the safety of the patient, we should certainly not neglect it in cancer of the pharynx.

With regard to the extent of the neck operation, here also there is some difference in practice. Some go to the trouble

of removing all the fascia, fat and glands from the whole of the side of the neck. This plan seems to me to be much safer than total neglect. But is such an extensive removal necessary? In nature, the infection from the pharynx seems to select a definite route—the carotid glands, and (or) the submaxillary glands, and those of the posterior triangle, to mention the regions accessible to the operator. That being so, it seems to me that if we confine our attention to those groups and to the connective tissue in which they lie, we shall be doing all that is necessary. I may note also, however, that retrograde extension of the disease may occur, and we ought therefore to carry our removal upward along the jugular vein towards the base of the skull.

Finally, I should like to add this to my logical arguments: it is by results that we must be guided. Consequently, we shall welcome the plan that gives us the best.

As to the part played by diathermy in the neck operation, we shall describe it in detail later on.

I proceed now with the various operations at our disposal, dealing first of all with what appears to be

ERADICABLE CANCER.

The Tonsil.—We shall take first of all epithelioma confined to the tonsil and the neighboring mucosa, excluding the tongue.

The disease seems to take origin usually in the body of the gland, probably in its crypts, where indeed, it may lurk unsuspected for a considerable time. How long that time may be, it is impossible to say. Cases occur, as we all know, in which the first sign of the disease is the appearance of enlarged cervical lymphatic glands, and in which a careful scrutiny of the pharynx fails to reveal any primary growth. In these circumstances, the tonsil of the same side, if removed and examined, will often reveal itself to be the seat of the latent cancer.

As a rule, however, the growth on the tonsil is plainly visible on inspection of the throat, and it usually manifests itself as an indurated ulcer on the surface of the gland. Sometimes the growing bulk of an epithelioma, starting inside the tonsil, leads to its extroversion in mass, very much like the tonsil itself when we are enucleating it with the guillotine, the cancer appearing on the surface as a cauliflower-looking mass.

(In these regions it is usual to describe epithelioma as occurring in two clinical forms: the papillomatous or cauliflower type, in which the main mass of the growth lies above the surface; and the flat or infiltrating type, which, burrowing in the deeper layers of the mucosa, is less conspicuous than the former. But it is the more malignant and the harder to extirpate of the two.)

From the tonsil the epithelioma readily spreads into the neighboring mucosa, invading the pillars of the fauces, the soft palate towards the uvula, and also sometimes passing on to the mucous membrane of the alveolus and into the adjoining part of the base of the tongue. Those two last extensions may convert an eradicable into an ineradicable case.

In operating for the removal of a discrete tonsil epithelioma, we are guided by what we have already said regarding the spread of the disease. Thus there are two fields of operation: that, namely, of the primary growth in the pharynx; and, secondly, that of the gland-bearing regions in the neck. In my own practice I prefer to operate upon the neck first of all, principally in order to ligature the external carotid artery so as to limit the hemorrhage during the pharyngeal operation, and to lessen the chances of serious secondary hemorrhage during the separation of the diathermy sloughs in the pharynx.

With regard to hemorrhage during the pharyngeal operation, it is often stated that diathermy prevents bleeding by sealing the vessels before they are divided, and that is true, but true only to a certain extent. Veins, capillaries and small arteries are sealed, but if an artery of moderate size is divided, and the external carotid has not been tied, it frequently spouts freely and requires a ligature, and it is often difficult to apply a ligature to tissues blanched, desiccated, and hardened by the coagulation.

As regards secondary hemorrhage during convalescence, this does not seem, if we can judge by the records, to be common, but I fancy that it occurs more frequently than we might suppose from the literature.

In any event, to tie a ligature round the external carotid artery during the operation on the neck is simple and easy, and it is well worth the slight extra trouble entailed.

The operation on the neck consists in the removal of the lymphatic glands and gland-bearing fascia of the submaxillary, carotid and posterior triangles of the neck. Our skin incisions, therefore, should provide access to those areas. Perhaps the simplest plan is to make an incision along the anterior edge of the sternomastoid muscle, with two subsidiary incisions, one upwards and forwards over the submaxillary region, and the third backwards over the posterior triangle. As a matter of fact, however, both of these latter areas can frequently be reached through the vertical incision. This incision is deepened until the carotid sheath is laid bare from the digastric muscle to the lower border of the thyroid cartilage, or lower still if need be. The sheath and the fascial extensions in its neighborhood are removed along with the glands, whether they are enlarged or not. If at all cramped for room, the operator should not hesitate to resect more or less of the sternomastoid muscle. In this dissection I usually employ the ordinary scalpel. The monopolar needle of Wyeth, however, could be employed.

(Wyeth's Monopolar Needle.—Wyeth uses an apparatus providing a high frequency current of sustained oscillation with an exceedingly high rate of alternation. The active electrode is a thin needle or a narrow blade. The incision is produced by an arc effect and the coagulation extends only to about one-tenth of a millimeter. It can therefore be used for skin and other incisions that are to be subsequently sutured. But although it seals the capillaries, the larger vessels are cut across and bleed as usual.

Wyeth's needle may be used with safety in the dissection of cervical glands and other delicate work. Its employment obviates the necessity of applying the ordinary diathermy terminal to the cut tissues.)

After scalpel dissection, on the other hand, raw edges of fascia and the bed of the removed glands, especially if these have been enlarged, should be touched with the diathermy electrode in order to prevent the infection of the wound with active cancer cells.

For the same reason, when we come upon an enlarged gland in the course of our dissection, it is advisable to puncture it in several directions with the diathermy needle so as to destroy the living cells within it before we proceed to excise it. The

shriveling up of the gland under the section of heat facilitates its removal, and the diathermy prevents the dissemination of live cancer cells should the gland be accidentally punctured.

After the neck has been cleared in this way, and the external carotid artery tied, the active diathermy needle may be plunged several times into the tissues under the angle of the jaw between the neck wound and the tonsil; or it may be inserted obliquely across the area: the idea being to seal the lymphatic vessels in this region.

("Block dissection" implies the removal of all the lymphatic-bearing tissues immediately draining the region of the primary growth. An ideal block dissection is seldom attainable in practice, and the area just referred to exemplifies one of the sections of a block that cannot conveniently be removed. But its lymphatic vessels can be, at least partially, sealed by diathermy puncture as just described.)

These various applications of diathermy lead to a certain amount of serous exudation during the first few days following the operation. For that reason, a large drainage tube had better be inserted along the whole length of the wound, emerging at its lower end. Otherwise the extensive wound may become septic.

The neck operation completed, we proceed to the removal of the primary growth in the pharynx. This may be done at the same seance as the neck operation, but it is often judicious to delay it for several days, particularly when the patient is frail and old, as so many of them are.

The most convenient method of removing the tonsil and its growth is to place the patient's head in the inverted position, using the Davis gag. The surgeon is seated at the head of the table. One precaution we must observe is that as diathermy is being used, anesthesia by ether must be avoided, as a spark from the electrode may fire the explosive mixture in the patient's air passages.

In proceeding to remove the growth, our first step is to coagulate the whole of its surface with diathermy, as that enables us to seize hold of it with tonsil forceps without any risk of detaching living cancer tissue and implanting it in any of the raw surfaces we may make later on.

The next step is what is called the "circumvallation" of the growth. By means of the sharp diathermy knife the growth is encircled by an incision carried through healthy mucous and submucous layers from a quarter to half an inch (about 1 cm.) outside the apparent limits of the epithelioma. In front, this usually results in the complete removal of the anterior pillar of the fauces. Next, curving round above the tonsil, we pass high into the soft palate close to the uvula. Below, the incision cuts through the tongue immediately below the tonsil fossa. Behind, the posterior pillar of the fauces will be sacrificed and it will be found that the incision will pass through the lateral wall and as far back as the posterior wall of the oropharynx. It is sometimes difficult to make the posterior circumvallate incision unless we use an electrode sufficiently curved to carry it over and round the projecting tonsil growth. (In the foregoing description, the terms, "above" and "below" refer to the pharynx as viewed directly. With the patient's head inverted these terms will be reversed.)

The circumvallation completed, it is a good plan to plunge the active knife deep to the tonsil from the anterior incision into the peritonsillar region in several directions, so as to coagulate the tissues here. By so doing we effectually prevent the squeezing of any of the deeper living cancer cells into the lymphatic vessels in our subsequent manipulations.

We have now completely isolated the growth by an impassable barrier of coagulated tissue and may proceed to its removal en masse.

This is done by seizing the isolated mass by the edge of the anterior encircling incision and dissecting the whole tonsil out of its fossa with the diathermy knife, proceeding slowly so that the process of coagulation precedes us everywhere as we progress. By doing so we can remove the whole epitheliomatous tonsil without any bleeding whatever. It may be remarked that this dissection is not as easy as it sounds. Not only are tough connective tissue bands frequently encountered, but the coagulation certainly makes them more resistant, and the sharp edge of the diathermy knife is necessary to divide them.

There is, we must not forget to mention, another method of using the diathermy current in the act of dissection. This consists in raising the amperage until sparking is freely pro-

duced between the electrode and the tissues. Then, by advancing the knife, an arc incision is made, and, the solid coagulation in depth we have described not being produced, the dissection becomes more rapid and easy. (In this method, the electrode does not come into actual contact with the tissues.)

Some operators may prefer the slow; others the rapid method. But the best plan probably is to become a slave to neither and to select now the one, and now the other, according to the exigencies of the moment.

It is to be remembered, however, that when we use the rapid, or arc method (and this applies also to Wyeth's needle), the coagulation obtained is much shallower in depth than when we employ the slow method, and in pharyngeal cancer, at least, a deep is preferable to a shallow coagulation for two reasons: first, because it minimizes the likelihood of leaving live cancer cells behind us; and second, because it widens the range of the sepsis-destroying action of the current. When the arc is flowing between the electrode and tissues most of the heat is dissipated in the air and on the surface of the tissues. When, on the other hand, the electrode is in direct contact with the tissues the heat, penetrating the latter with but little loss, exercises its full action in depth.

It is said that bleeding is more apt to occur with the slow than with the rapid method. I can only say that has not been my experience.

Having separated the anterior half of the tonsil from its fossa, the knife is next passed in an upward direction towards the encircling incision in the soft palate, and the upper pole of the tonsil is dissected free. This quite frequently entails the destruction of the corresponding side of the velum palati.

We turn next to the incision through the tongue and free the lower pole of the tonsil.

There only remain now the posterior connections to be divided, and these are most easily reached from the front, the partially separated tonsil being held away from its bed by tonsil forceps.

The whole mass having been thus removed, there is left an extensive raw surface from the anterior faucial pillar in front to the posterior wall of the oropharynx behind, and from the surface of the tongue to the base of the uvula.

The final act of the operation consists in submitting the whole of this area to the action of the coagulating terminal. For this, a blunt electrode is the most suitable. The tissues, it may be observed, are already desiccated and coagulated, but our final application carries the coagulation deeper. The amperage of the current has to be increased if a larger electrode is used, the electrode being held in contact with each segment dealt with for as many seconds as are required to blanch the tissues.

Care should be taken, however, not to use too strong a current, for then, as Nagelschmidt points out, the coagulation will be limited to the surface of the tissues. To obtain a deep effect the current should be mild and the electrode should remain in contact for ten or fifteen seconds, until, with some rapidity the characteristic blanching appears.

Even then the depth of penetration of coagulation is not so very great, not nearly so great as in dead tissue, as the circulating blood acts like the radiator of a motor car and cools down the exposed area. While that is true, I confess, however, that in applying the electrode to the lateral pharyngeal wall behind the tonsil fossa, I have a lively sense of the proximity of the internal carotid artery and the internal jugular vein, and leave it in contact at this area for a shorter period than in front. In this our final act, particular attention should be paid to the adjoining region of the tongue, of which we ought to include a generous portion in our coagulation.

Here also I may refer to an additional precaution. In one of my cases in which recurrence took place, the base of the tongue was the first to show the new development. I noticed that the affected portion of the tongue was that which, when the mouth was closed, had come into contact with the original growth in the tonsil. So I concluded that we had to deal with a contact infection. It would seem, therefore, advisable to apply the active terminal to such surfaces on the tongue and uvula as habitually come into contact with the epithelioma.

As we have seen, the raw surface left by this excision is extensive, and as it interferes to some extent with swallowing, an esophageal feeding tube may be of advantage. Even with great losses of substance, however, the patient soon acquires the knack of swallowing.

Postoperative Treatment.—Two remarkable features characterize the patient's postoperative condition; first, shock is often absent, and second, pain is slight compared with the extent of the wound.

Pain, however, is occasionally experienced, but it is much less severe than after the cutting operation. Its absence is probably due to the coagulation necrosis of sensory nerve endings deep to the actual operation layer. Probably the same circumstance, together with the hemostasis and the general warming of the body, accounts for the frequent absence of shock. I use the words "frequent absence" because, although it is sometimes said that shock is always absent, that has not been my uniform experience.

In this connection we may note that a rise of temperature to 101 or 102 degrees F. (38 or 39 degrees C.) usually occurs during the first twenty-four hours after the operation. I have heard it said that death has occurred after a prolonged exposure to surgical diathermy, but I have not seen any serious after results myself.

Again, it is noteworthy that in spite of sloughs and necrosed tissue, which undoubtedly are themselves the seat of bacterial invasion, as they frequently become fetid, the living tissues beneath them are not infected, and there is little or no rise of body temperature after the first day, and no evidence of toxemia.

The local treatment of the mouth consists in keeping it as clean as possible by frequent syringing with a Higginson syringe and some mild antiseptic. If the sloughs are at all fetid the throat is sprayed with weak eusol or hydrogen peroxid. If as they separate they hang in loose tags and irritate the pharynx, it is advisable to remove them gently with forceps, or if they are tough to clip them off with scissors. Forcible detachment by pulling should be avoided, as bleeding may be started by so doing.

The wound in the neck, having been more or less exposed to diathermy, and containing necrosed tissue, discharges serum freely for a week or more, and quite frequently the discharge becomes purulent, but, drainage being provided, no serious septic infection takes place. (I should mention that, as it is dif-

ficult to sterilize the handles of the electrodes, at the operation they are wrapped in a sterilized towel while being used. The metal electrodes can be sterilized by boiling.)

The sloughs in the pharynx begin to separate about the fifth day, the more bulky coming away during the next fortnight, leaving a whitish adherent layer which gradually thins out and disappears, perhaps being partly absorbed, during the next four or five weeks. Epithelialization is rapid and is usually completed about eight or ten weeks after the operation.

When the epithelium fails to cover the whole of the raw surface, and an island of raw granulation tissue is left in the center of the operation area, one should regard the spot with some apprehension, as this is just the appearance presented when our operation has failed to remove the whole of the epithelioma. As a rule, however, it consists merely of simple granulation tissue that a little patience will see covered over. If there is any doubt, it is best to remove a piece for microscopic examination and to coagulate the patch with diathermy.

Epithelialization is followed by a contraction of scar tissue which is frequently so pronounced as to narrow the isthmus of the fauces by the dragging up of the side of the tongue towards the soft palate. The scar contraction may be so great as to limit the movements of the jaw and to render subsequent examination of the pharynx difficult without the use of the laryngeal mirror. The amount of scar contraction, however, depends upon the extent, the depth and to some degree also upon the situation of the area diathermized. In the floor of the mouth, for example, if the neoplasm has been small and nonadherent, the scarring will be slight and the contraction negligible. The difficulty in opening the mouth after the tonsil operation is probably due to scar contraction of the internal pterygoid muscle.

Accidents and Complications.—Some operators report the onset of edema of the mucosa around the wound during the first few days after operation. This I have noticed only once or twice, and then it was trifling.

As a result of diathermy puncture of the structures intervening between the neck wound and the tonsil, I have once seen a communication form between the pharyngeal and the

neck wounds during the separation of the internal sloughs. It quickly closed, however.

Severe secondary hemorrhage from a vein took place in one case, necessitating the application of the tonsil clamp for several hours. But it did not persist and the patient recovered.

There have been no deaths from hemorrhage, an escape we may attribute to the ligature of the external carotid.

Soft Palate and Uvula.—For cancer beginning at or near the middle line of the soft palate, or in the uvula, the entire soft palate should be removed.

This is preferable to removing only that section which contains the growth, as it not only affects a wider clearance of the disease but it enables the dentist to block the opening subsequently with an easily made obturator.

The soft palate may be removed by the diathermy knife in one of two ways. In the first, it is divided on the one and then on the other side of the uvula from velum to near the hard palate. The central segment thus demarcated, containing the uvula, is removed by a transverse incision close to the edge of the hard palate, and the lateral pieces are then detached by incisions close to the lateral wall of the pharynx on either side. In this operation the diathermy knife may traverse the epitheliomatous area, but there is no risk of dissemination, as all the cancer cells approached by the electrode are destroyed by heat.

In the second, the diathermy knife, entering the velum palati just above the tonsil of one side, passes upwards to near the edge of the bony palate, then transversely across the soft palate to the opposite side and so downwards to emerge at a point corresponding to that of its entrance. The entire soft palate is thus removed in one single piece.

Whichever method be adopted, the tonsils also should be removed with the diathermy knife, as recurrence is apt to appear in one or other of them. (This hint I owe to Sir St. Clair Thomson.)

Partial removal of the soft palate on one or other side is sufficient when the growth is situated laterally or when it has extended to the soft palate from the tonsil or faucial pillar.

The cervical operation in these cases depends upon the situation of the primary growth. If it is at or near the middle

line, both sides of the neck should be cleared. If it is lateralized, a unilateral clearance should suffice.

Before leaving the palate, I may take the opportunity of explaining why I remove more or less of the structure in its entire thickness instead of only the mucous and submucous layers in and around the growth. My reason has been that when the growth is a sarcoma within the substance of the palate, complete removal is obviously indispensable, and when it is an epithelioma of the surface, if we leave the deeper parts alone, we run the risk of leaving behind the deeper extensions of the growth. It is possible that, in the latter case, my practice is unduly radical. Here, however, as in other directions, I am ready to be converted by results.

I may add that an obturator fitted by the dentist to a tooth plate to fill the gap in the palate is sufficient to deprive the speech of any nasal quality.

The Epiglottis.—Epithelioma frequently spreads to the epiglottis from the aryepiglottic fold or from the vallecula. In such cases the disease is obviously not easy to excise. As a rarity, however, it starts in the epiglottis and has not spread beyond it by the time the case comes under observation. When so limited, eradication by diathermy should be attempted.

To excise the appendage through the mouth must be difficult, whether we are operating with the Davis gag or through the tube spatula. On the other hand, diathermy destruction of the cancer in situ will be more easily accomplished.

If, however, deliberate excision is preferred, then the epiglottis should be approached through such an external opening as is provided by subhyoid pharyngotomy, as the appendage is thereby more clearly defined and manipulated.

(In all diathermy operations in and about the larynx provision should be made by the performance of a preliminary tracheotomy for the onset of postoperative edema.)

Pillars of the Fauces.—Epithelioma of the anterior faucial pillar without apparent involvement of the tonsil is removed by an operation like that for a tonsil growth, and the tonsil should also be removed. In the neck operation, particular attention should be paid to the submaxillary as well as to the carotid triangle, as it is in the former that the glands directly

draining the anterior pillar and adjoining floor of the mouth are situated.

I have never seen epithelioma confined to the posterior pillar of the fauces, but it is frequently enough invaded from the tonsil and is removed along with it in the diathermy operation.

The Tongue.—One of the consequences of our familiarity with major diathermy operations in the pharynx has been that cancer of the tongue is nowadays frequently referred to laryngologists. But as we are being favored today with a contribution on the subject by Norman Patterson, whose experience in the matter is much greater than mine, I do not propose to deal with it.

Posterior Wall of the Oropharynx.—Epithelioma starting in this situation is rare, and most of the cases one sees are really instances of extension, usually from the hyoid fossa or from the laryngopharynx (postcricoid region).

The diathermy removal of a small primary epithelioma of the posterior wall of the oropharynx by the methods of circumvallation and undercutting would be quite practicable.

The mucous and submucous tissues in this region are thin, however, and early fixation to the underlying vertebral body is the rule. In that case, local diathermy coagulation without formal excision would be necessary. (We deal with the treatment of epitheliomatous invasion of bone later on.)

The Introitus Laryngis.—We are now approaching extrinsic cancer of the larynx, in which are included cancer of the arytenoid region, of the aryepiglottic folds and of the pyriform fossa.

In the past this has been a melancholy chapter of laryngology, and even today, with all our recent improvements, it remains unsatisfactory. Nevertheless, some advance has been made.

Until within the last ten or fifteen years surgery, confronted with epithelioma in these regions, has had to acknowledge itself without resource. In some cases, no doubt, particularly when the growth is confined to the interior of the larynx, but also when its extensions on the outside have not involved the pharyngeal mucosa to an undue extent, it is possible to remove it by laryngectomy. But in most cases operation has been out of the question.

Wilfred Trotter of London, however, by the method of approach he terms "lower lateral pharyngotomy," has enabled us to expose the diseased area from without and to excise it, at least in suitable cases.

Broadly speaking, the operation consists in the exposure of one side of the pharynx through a lateral incision in the neck, carried down to the cartilages of the larynx, the ala of the thyroid and the cornu of the hyoid being partially removed. The pharynx, exposed in this way, is opened by a longitudinal incision, and that gives access to the introitus laryngis and epiglottis, and lower down, to the laryngopharynx. Trotter then removes, with scissors, as much of the larynx as is necessary. Those who favor diathermy will employ the diathermy knife instead of scissors, for the reasons already given.

The operation also provides us with access to the hyoid fossa. But in cancer of that region, if we are operating on the same side of the lesion, the separation of the hyoid and thyroid must be effected with caution, as the underlying pharyngeal mucosa, infiltrated as it is with the disease, is easily torn. If this accident is avoided, the diathermy knife can be used, under the guidance of palpation, to excise or to destroy the neoplastic area from without. Frequently, access to the area is more conveniently secured by operating through the opposite side of the neck.

Needless to say, the operation is one of some magnitude, and the immediate mortality has been higher, at least in my hands, than that of the other operations we have been considering. Further, it is difficult to define exactly the extent of the disease before we operate, and it may prove to be greater than we anticipated. That being so, the operation should be reserved for cases in which the foregoing examination clearly reveals the situation and extent of the growth to be limited.

The Postcricoid Region.—Epithelioma of this area seldom comes to our notice at a stage when we may hope for eradication. Sometimes, however, as when the growth is confined to one side of the laryngopharynx, or when, although encircling the lumen, its vertical extent is short, it may be excised through the lateral pharyngotomy opening, the ulcerated area being removed with the diathermy knife or with the endothermy needle.

Holmgren, by the way, reports a case of what was evidently a cancerous growth of the papillomatous type removed, with the aid of the directoscope, by a snare from the hypopharynx of a woman, aged 61, the wound being diathermized. The tumor lay on the posterior wall. The patient was alive and free from symptoms at the time of writing, twenty months after the operation, although a small diverticulum had formed.

Laryngofissure for removal of intrinsic cancer of the larynx, that is to say for a growth confined to one vocal cord or ventricular band, has become so safe and successful a procedure that the use of diathermy to effect the removal of the disease area after its exposure seems like a redundancy. Moreover, even the warmest enthusiast for diathermy will hesitate to employ it for that purpose: first of all, because the thyroid cartilage may be necrosed by the diathermy as the soft parts are being dissected off it; and secondly, because a bulky diathermy slough in the interior of the closed larynx may set up bronchopneumonia.

But while for these reasons we prefer to excise the soft parts with the ordinary scalpel, the raw surfaces left may be touched with the electrode in such a way as to seal the lymphatics and to stop bleeding. The neighborhood of the cartilage should be avoided.

Complete diathermy removal would be possible without risk of cartilage necrosis, if access were made through a window in the thyroid cartilage, as in the operation of Lambert Lack. Or, here again, the endothermy needle of Wyeth, which confines coagulation to a narrow line on either side of the track, might be tried without risk of damaging the cartilage.

It appears that peroral diathermy coagulation of early intrinsic carcinoma has been performed through the direct tube or under suspension, but I find some difficulty in looking upon this as a serious effort to extirpate cancer of the larynx, particularly in view of the safe and certain removal by laryngofissure.

When the entire larynx is being removed, as for bilateral intrinsic cancer, Wyeth advises that its connection with the pharynx should be severed with his endothermy needle, as that instrument produces much less sloughing than the diathermy knife. I have not yet had any experience myself of Wyeth's

needle, but if it comes up to expectation, there can be no objection to its employment at this stage of laryngectomy. On the other hand, I have always rejected the diathermy knife for the cutting of the pharyngeal mucosa, because of the fear that the sloughs would prevent the sutured gap remaining water tight.

In concluding this section we may observe that when eradication has been, to all appearance, successfully accomplished, the case should return for periodical examination for a prolonged and even for an indefinite time. It frequently happens that a small local recurrence appears, which, if destroyed by a timely diathermy coagulation, can be prevented from spreading. In one or two of my cases in which this was done the recurrences finally ceased. Thus we can, as it were, keep up a pursuit of the disease until it is extirpated. Furthermore, as one of my cases shows, a period of as long as five years may elapse before recurrence takes place.

INERADICABLE CANCER.

This variety is unfortunately much more often met with than the other. Taking my own numbers I find that the proportion of eradicable to ineradicable cases submitted to diathermy is as one to four. And this ratio underestimates the difference, as it omits the cases that were too far advanced for treatment.

The reasons for this deplorable state of matters are known to all of us, and they are not entirely removable. It is true, no doubt, that if practitioners were trained to refer all cases of pharyngeal infiltration or ulceration promptly to the expert, many more would reach us early enough to justify an attempt at complete removal. It is a significant fact that ten out of the twelve cases of apparently eradicable, or at least early cancer appended to this paper were seen in private, two of them being medical men. On the other hand, during the same period of time upwards of fifty ineradicable cases were treated, and of these only five were private patients—a telling contrast.

But there is another factor in the situation, a factor not so easy to combat, and that is that the disease is often far advanced before the patient himself becomes aware of anything amiss. For, apart from the rare concealed cancer of the tonsil to which we have referred, it is a common occurrence for the

disease to grow to implicate a large area of the pharynx and even to infect the cervical glands, before attracting any attention.

That being so, we shall, I fear, continue to receive many of our cases too late for radical treatment.

In these regions we may consider the cancer to be ineradicable when

1. It has penetrated deep into the base of the tongue—a frequent occurrence;

2. When it has become adherent to bone, as in the hard palate, the mandible, or the bodies of the vertebræ;

3. When it is situated in the glossoepiglottic fossa;

4. When, in the hyoid fossa, the introitus laryngis, or the laryngopharynx, it is so extensive as to prohibit removal;

5. When the infected cervical glands are large, adherent and matted together;

6. When it is situated in the esophagus.

With regard to the cervical glands, if, albeit enlarged, they are solitary or few, and nonadherent, they should always be removed as described above. For although, as I shall show later on, my experience corroborates that of Professor Schmiegelow, and of Clark and Berven, that when infected glands are removed, recurrence always takes place, yet we have to remember first, that enlarged glands are not always epitheliomatous glands, and, secondly, that if they are epitheliomatous, their removal will retard the progress of the disease and obviate much of the patient's discomfort.

In many cases, even when the primary growth is or seems to be ineradicable, the cervical glands are still small and discrete. If so, they should be removed, and the external carotid artery ligatured, just as if the primary growth were eradicable. For, as I hope to show later on, a prolonged interval of freedom from the disease may thereby be secured.

If, however, the glands are so matted together that they cannot be enucleated, or if they are multiple and can be felt to be enlarged as far down as the supraclavicular region, then any attempt to remove them will be futile. In these circumstances, however, they may still be treated by diathermy puncture in situ.

Sometimes we embark upon a neck dissection with the expectation of being able to remove the glands, and then their exposure shows them to be so adherent to the great vessels, the muscles, and each other that we are forced to abandon our plan. In that case the diathermy needle inserted repeatedly into the substance of a gland as it lies in a wound reduces its size and seems to restrain its subsequent enlargement for a considerable time. (In making these punctures care should, of course, be taken to avoid the great vessels.)

When, however, the diagnosis of adherent glands can be made before the neck operation is attempted, the needling of the glands does not necessitate a large, open dissection. All we require to do is to make a short opening through skin, superficial, and deep fascia, until close on to the glandular mass, and then to pass the needle into it in several directions, being careful not to approach too near to the great vessels. Diathermy puncture of the glands was first suggested and carried out by my colleague, W. Stuart-Low.

It is advisable, if the gland appears prominent at one place under the skin, to make the little incision some way from the projection and to approach the gland obliquely. For, if we incise the skin directly over the prominence, enlargement of the gland at a later date may lead to it breaking through the skin at the site of the incision. The small wounds thus made can be sutured without being drained.

In the later stages when the enlarged glands are beginning to soften, no attempt should be made to treat them with diathermy, as the wound will remain open and become septic, and we shall only add to the patient's miseries.

So much for the treatment of the glands in ineradicable cancer. We now turn to the diathermy treatment of the primary growth. Naturally, the details vary with the locality and extent of the neoplasm, so we shall follow the same plan we adopted in the first section and take each locality in turn.

But here also we may start with a brief exposition of the principles that guide us in our treatment.

Before the discovery of surgical diathermy, cancer in the pharynx, or for that matter cancer anywhere in the body, when it passed beyond the eradicable limits, was left alone, for the very good reason that interference, as by partial excision,

curetting or cauterizing did more harm than good. Such partial removal seemed indeed only to irritate the growth and to quicken its progress. The causes of this disconcerting sequela are that a partial operation leads, first, to the grafting of living cancer cells upon the healthy tissues around; and, second, to septic invasion of the area and so to increased vascularity and stimulated growth.

There is a close connection between malignancy and sepsis. Cancer itself varies in virulence, as we all know, in accordance with some factor, the nature of which we can only surmise. Intrinsic epithelioma of the larynx, for example, is a slow growth of low malignancy; while, on the other hand, lymphosarcoma of the pharynx is of rapid growth and high virulence. But while the cause of such inherent disposition is obscure, one factor in the variable behavior of different cancerous growths is well known, and that is that when a cancerous area becomes infected with sepsis, and owing to its low resistance to septic organisms it quickly does become infected when exposed to them, the growth takes on a much more rapid progress, no doubt owing to the increased vascularity induced by the infection.

Now cancer, or at all events epithelioma, whether of the pharynx or of the exposed regions of the larynx or of the esophagus, being an open ulcer almost, if not quite from its inception, becomes infected with the bacteria of the mouth at an early stage of its existence, particularly if teeth are still present. And in the natural course of events it remains septic, the sepsis growing ever worse until its termination.

An early and frequent indication of this complication is to be found in the simple enlargement of the glands at the angle the jaw, and so we may say that the septic invasion of the glands frequently precedes the neoplastic invasion. Finally, in the later stages of the disease it is the sepsis that is responsible for the softening of the glands, their suppuration and their forming open, discharging sores on the neck.

The consequence of this extensive and persistent septic invasion is that it is usually some septic event that gives the patient his *coup de grâce;* either general septicemia, or septic pneumonia, or general asthenia from the combination of pain, insufficient nutrition, repeated hemorrhages and septic intoxica-

tion. Thus the intrusion of sepsis, besides adding to his sufferings, shortens the patient's life. It is said that the average duration of an untreated case of cancer of the pharynx from first to last is two years. I should myself put it as much less than that—at about six months from the time when it is first discovered.

In the production and maintenance of sepsis the teeth undoubtedly play a predominant part, for when cancer arises in edentulous people it not seldom remains aseptic, and it is then much more slowly progressive. One such case that I saw some years ago of a papillomatous looking epithelioma of the laryngopharynx, in an edentulous woman, remained alive for more than ten years after the discovery of the growth.

Thus part of the problem before us is, assuming that the cancerous area cannot be extirpated, how to make and keep it aseptic in order to prolong our patient's life in comfort.

Obviously, our first duty is to reduce to the minimum the pathogenic flora of the mouth by the removal of all teeth. This precaution ought, as a matter of course, to be taken in all cases of the cancer of these regions, whether apparently eradicable or ineradicable, unless, of course, the disease is already too far advanced for any treatment. Curiously enough, it is said, and my own experience bears out the opinion, that a mouth that has long been edentulous contains fewer bacteria than one from which the teeth have only recently been removed. Perhaps this is no more than we should expect, but it is an unfortunate fact from our point of view.

One of my own cases exemplifies this circumstance (Case 4 appended). It was that of a woman who had lost all her teeth many years before her epithelioma appeared, and I remember being struck by the pale, quiet, inactive look of the growth and by the absence of redness and infiltration around it. Progress in the case was strikingly sluggish and painless, for although recurrence appeared sixteen months after operation she did not succumb until two years and three months later.

Nowadays, as it happens, we are able in many cases even of ineradicable cancer of the pharynx, to supplement the precautionary cleansing and clearing of the mouth by a sterilization of the cancerous area itself. For diathermy has this power

to a notable degree. That is to say, by diathermy we can not only coagulate and destroy a cancerous growth *in situ*, but, even when we fail to destroy it utterly, we can reduce and even abolish the septic element in it, at least for a time.

Diathermy possesses therefore two valuable properties: it kills all cancer cells within the radius of its coagulating action, and beyond that radius it sterilizes living tissues of septic bacteria. That being so, its application, we infer, must be free from the risks attendant upon a partial operation with ordinary surgical instruments. And that our inference is correct is borne out by our experience of the use of diathermy as a palliative in ineradicable, in what indeed in the prediathermy days was called "inoperable" cancer.

One precaution, however, should be kept in mind. In selecting an apparently ineradicable cancer for diathermy treatment, we ought to assure ourselves beforehand, as well as we can, that, although ineradicable, it is of such a position and extent that we can apply the diathermy to its entire area. To diathermize one part and to leave another part of the growth wholly uninfluenced probably does more harm than good.

We now proceed to consider in detail the diathermy treatment of ineradicable cancer of the different regions.

The pharynx, including the tonsil, the faucial pillars, the palate and the base of the tongue.

Having dealt with the cervical glands in the manner best suited to the particular case, and having ligatured the external carotid artery if possible, we make a direct application of diathermy to the primary growth, coagulating it in its entire area, and as far as possible, in all its thickness, no attempt being made to excise it. For this purpose, the pointed button or disc electrode is perhaps the most suitable, and the application should be made slowly, deliberately and thoroughly. Most operators, including the writer, having coagulated the ulcer as fully as possible, proceed to remove the dead tissue with the ordinary curette in order to expose the underlying noncoagulated layers to successive applications of the diathermy. I have already given my reasons for preferring circumvallation and excision to coagulation *in situ* and curetting ineradicable growths, and to these reasons I must also add the drawback mentioned by E. P. Cumberbatch that by curetting we are apt

to detach pieces of what is still living malignant tissue which may infect distant regions.

But while these reasons apply when, as in eradicable cancer we possess the alternative method of excision which is free from those drawbacks, in ineradicable cancer we are compelled to curette, as no alternative method is at our command. Cumberbatch's warning, however, should lead us to be careful to curette only such tissue as is manifestly coagulated and to proceed by small stages.

(In order to obviate those perils I have recently had a curette-electrode made. By using the back of the instrument we can coagulate the tissue and then, turning it over, we can curette with the current still passing.)

In this procedure, when the diathermy is applied over a bony surface, as when an epitheliomatous ulcer is adherent to the bone of the mandible or hard palate, no hesitation need be felt in destroying the tissues down to and including the surface of the bone. The superficial layers of bone are killed by the heat and a sequestrum subsequently forms and exfoliates; but the process, like that of the shedding of sloughs of the soft tissues, takes place aseptically and no ill effects follow. If then these layers contain epitheliomatous extensions they perish along with the bone.

The results we obtain from the use of diathermy in an apparently ineradicable growth of the pharynx depend, naturally, upon the depth of penetration of the diathermy, and that often depends upon the accessibility of the growth. In the oropharynx, we are able in most cases to effect a complete coagulation of the whole area. If any difficulty be experienced, as, for example, in reaching the most posterior parts of the growth, the cheek of the opposite side may be split from the angle of the mouth downwards and backwards to the masseter muscle, as this gives us direct access to the affected area. The cheek incision heals rapidly and leaves an inconspicuous scar.

The most difficult area to diathermize is the base of the tongue, and indeed anything like a systematic layer coagulation in this quarter is practically always out of the question. But I have succeeded on several occasions, by a repeated plunging of a diathermy needle deep into the cancerous infiltration, in causing a decided arrest, and even a retrogression of the disease

in the tongue. Diathermy puncture produces an aseptic necrosis along and around the needle track, followed by the formation of scar tissue.

If we have been able to destroy the ulcer entirely without exposing any bone, complete epithelialization of the surface may occur, and the disease may be arrested for quite a long time, with its dysphagia, pain and salivation abated or abolished. In one of my cases, which was looked upon and treated as ineradicable, the patient, a medical man, had a respite from his disease for two and a half years, during which time he was able to carry on his practice. (Case 7.) Thus our diagnosis of "ineradicable" may sometimes require modification.

The contrary is, of course, also true; we sometimes set out with the confident expectation of extirpating the disease, only to find at the operation that, although much of it can be excised, we cannot do any more than a layer coagulation in certain parts of the growth. (Instances of both of these errors will be found in the case records appended.)

In cases with bone exposed by the diathermy, epithelialization frequently advances as far as the raw bone surface.

In the more advanced cases, epithelialization is only partially effected. Yet even in advanced cases, when we have been able to make a satisfactory application, the ulcer cleans up, fetor is abolished, pain is relieved and may even for a time entirely disappear. This welcome relief lasts for from four to six weeks or longer, and then the symptoms return, rather suddenly, probably because the cancer, beginning to grow again, has received a new infection of sepsis.

On the return of the symptoms the diathermy application may be repeated, and at a later date repeated yet again. In this way patients with not too advanced though ineradicable carcinoma may be rendered so comfortable that for a time they may even be able to return to their place in the world.

Sooner or later, however, there comes a time when the combination of cicatrization and the extending neoplasm so limits the opening of the mouth that free and open access to the pharynx for inspection and treatment is no longer possible. Or we may find that our efforts are failing to relieve the pain and to diminish the ulcerated area. When that stage is reached it is time to abandon the treatment.

Ineradicable cancer of the pyriform fossa and of the introitus laryngis is in some cases most readily approached by suspension. Or the tube spatula may be employed, the diathermy electrode with a long, fine handle being used. When diathermy is being applied one should, as a rule, avoid switching on the current until the electrode is in actual contact with the tissues, and it should be switched off again before removing the electrode from its contact. But it is when operating through a metal tube that this rule must never be broken, as otherwise the current may short-circuit to the direct tube and produce unnecessary burns. In such cases also, and particularly when we are operating round a corner, the structures in the way can be protected by covering them with a glass or vulcanite shield on a long handle, or with a piece of thick rubber tubing held in position by long pressure forceps.

The remote situation of these growths, however, and the readiness with which they bleed when the apparatus of approach is inserted, generally prevents anything like a satisfactory and complete diathermizing of their entire field. Nevertheless, the attempt is occasionally worth trying.

In most cases of postcricoid cancer the disease is definitely ineradicable, as it always is in cancer of the esophagus, and all we can hope for is to destroy the growth sufficiently to allow swallowing to be resumed. For this purpose, most of us have been using a cylindrical electrode at the end of a long, slender handle, passed through the esophagoscope and then pushed, while active, through the growth, keeping as near as possible to the lumen of the esophagus. Sometimes quite a gratifying measure of success is obtained, but there is a danger of penetrating the esophageal wall either at the moment or by the subsequent sloughing, an accident almost necessarily fatal. Recently in order to avoid this catastrophe, several ingenious electrodes have been devised, such as that of A. J. Wright of Bristol, which is passed inert through the lumen of the growth and then withdrawn active, thus diathermizing the growth only as it is being withdrawn. Other ingenious contrivances are doubtless known to you.

At the same time, it is legitimate to ask whether diathermy is likely to displace radium in the treatment of cancer of the esophagus. I am inclined to doubt it. Diathermy is, perhaps,

easier to apply, more certain, and, when carefully handled, more precise than radium, but in the esophagus it is undoubtedly more dangerous and probably not so efficacious. Nevertheless, again we must leave the decision to the future, looking for it in our results.

In ineradicable cancer of the larynx I have so far refrained from using diathermy at all, the reason being, that I am afraid lest perichondritis should be set up by the diathermy and the patient's chances of comfortable existence jeopardized. Indeed, his fear has been responsible for the reluctance with which diathermy has been used even in the milder and earlier forms of cancer of the larynx. I note, however, that Professor Schmiegelow has used it in laryngeal carcinoma without any deleterious after-effects.

In concluding this section, I must not omit to mention that diathermy has been used, under local anesthesia, for a growth in the trachea, just above the bifurcation, by Bowing and Vincent.

Leaving now the subject of the different local applications of diathermy in the pharynx, larynx, trachea and esophagus, I should like to advert for a moment to some instances in which the remedy has disappointed me.

Its use in epithelioma of the pharynx is, we may say, established; at all events, for the time being, and until we are furnished with something better. On the other hand, in some of my cases of sarcoma, more particularly in the highly malignant lymphosarcoma of the tonsil and base of the tongue, it has proved to be much less serviceable than radium. In one case, in particular, the application of diathermy with the object of destroying the primary growth was followed by severe pain, and it failed entirely, to all appearance, to inhibit progress. Upon this type of growth, so far, at least, as its local existence is concerned, radium seems to exercise an almost specific action.

No one who has ever seen the almost miraculous melting away of sarcoma of the pharynx under the influence of radium is ever likely to forget it, even although it does not seem to prevent the occurrence of metastasis. Much the same effect, as a matter of fact, is sometimes obtained from the use of deep X-rays.

On the other hand, in the class of parotid tumors, which we occasionally encounter in the palate and tonsil region, I have found diathermy superior to radium (an illustrative case is reported below.)

Still dealing with the subject of the limitations of diathermy, I should like once more to emphasize the importance of stopping diathermy, or of refraining from its use when the neoplasm is in its later stages, when ulceration is deep and extensive and the glands are large, adherent and soft. At this period any operative measure is to be avoided, as the local recuperative powers are so seriously impaired that healing of the wounds we make will not occur, and operation will only add to the patient's miseries.

Comparison of Diathermy With Radium and Deep X-rays.—We have already made several allusions to the relative merits of diathermy and radium, but there are still one or two matters upon which I should like to say a word.

There are some authorities who favor the use of radium so strongly that they are demanding that it should be employed in preference to surgery even in eradicable cases of cancer of the pharynx, and they claim that the results after radium in these cases are as good as or better than those of surgery.

This seems to be a high claim, but, if the surgical results referred to are those of prediathermy days, it is not so astonishing as it seems, since these results were nothing to boast about. But if we compare the results of diathermy treatment with the results of radium treatment, the claim for radium will be less easily substantiated and more reluctantly granted, for all who have had experience of diathermy are unanimous in agreeing that its results are much better than those of former days. Douglas Harmer, for example, says that "since diathermy has been employed, the number of cures that have been obtained has been almost double."

In the debate upon this question one or two matters demand our attention and consideration.

The great argument in favor of diathermy in eradicable pharyngeal cancer is that we are acting logically from sound pathologic data. We destroy the primary growth and seek to eliminate also those areas and structures where secondary deposits occur.

With radium, on the other hand, we are still, or so at least it seems to me, not quite so sure of our fundamentals. Radium undoubtedly kills neoplastic cells more readily than it does normal cells. (Diathermy, by the way, is said to do the same.) But can we ever feel assured that all the neoplastic cells in a radiated field have succumbed? Is it not probable that some will escape to carry on their evil work? Besides, there is some suspicion that the neoplastic cell is able to acquire a resistance to radiation.

There is another factor to be considered. It is difficult to make a widespread insertion of radium in tubes, needles or seeds without rendering septic the tissues in which they lie. Now, for reasons we have already fully dealt with, we cannot look with indifference upon an additional invitation to sepsis to invade a cancerous area.

Finally, we may ask, what is to be done with the cervical gland-bearing areas when we radiate a primary pharyngeal carcinoma? Are we to remove them by block dissection? Or are we to submit the whole region to the action of radium? Or are we to ignore all that pathology teaches us and to leave them alone?

In this connection I venture to utter a protest against the notion that to apply radium at intervals in a band around the neck provides in any way an effective bombardment of the gland-bearing areas. The fact is that rather loose ideas and practices are becoming prevalent at the present time and should be disavowed by all sensible men. When, for example, tracheal tumors are treated by an application of radium to the manubrium sterni, one is irresistibly reminded of folk medicine and the wearing of charms and amulets.

Such extravagances, however, should not blind us to the real value of a remedy. And I hasten to add, lest I be misunderstood, that the respective values of diathermy and radium can only be settled in one way: by their results namely and not by *a priori* reasoning. Let us therefore await findings before finally deciding.

With regard to deep X-ray therapy less skepticism may be entertained, because the value placed on this method by its exponents is not so high as that credited to radium. They are content to restrict its use to inoperable and ineradicable cases.

To its valuable alleviating properties in these cases, all of us, I am sure, can bear witness. It undoubtedly does mitigate the distressing symptoms of the later stages of cancer of the throat.

Finally, let us remember that we frequently are able to employ radium and X-rays in combination with diathermy, with great benefit. Thus, an adherent carcinoma may be loosened by the action of radium, and then eradicated by diathermy. Again, as Case 8 appended shows, if the insertion of radium is followed by septic infection, diathermy may be resorted to for its elimination. And there are other directions in which these agents can be used together which will occur to you. My appeal for caution regarding the claims made for radium is, I would have you note, an appeal for caution only, and by no means an expression of skeptical prejudice.

CASES.

1. Female, 64. Epithelioma of soft palate.

Jan. 16, 1919. Diathermy removal of entire soft palate and tonsil.

Feb. 18, 1919. Healed.

June 29, 1925. A small recurrence (apparently) in the scar destroyed by diathermy.

No further recurrence on Jan. 27, 1928—i. e., nine years since first operation.

2. Male, 55. Epithelioma of right tonsil, anterior faucial pillar and angle between pillar and tongue.

July 12, 1921. Operation on neck and diathermy removal of pharyngeal growth.

Oct. 17, 1927. Died. Postmortem showed recurrence in tongue without extension to fauces, and a mass of growth in the lumbar spine compressing the cord. No recurrence in the neck. No sign of recurrence for five years after operation.

3. Male, 59. Epithelioma of upper pole of left tonsil, extending on to posterior faucial pillar. Enlarged, hard gland at angle of jaw.

Feb. 26, 1923. Neck operation. Enlarged gland removed along with two inches internal jugular vein to which it was attached.

June 26, 1923. Growth in pharynx removed by diathermy.

Feb. 21, 1924. Recurrence in base of tongue.

April 4, 1924. Secondary growths in mediastinum.

Died.

4. Female, 70. Epithelioma of left tonsil, edge of tongue and soft palate. Fixed. Soft enlarged gland at angle of jaw.

June 27, 1923. Neck operation with ligature of external carotid followed by partial diathermy removal of pharyngeal growth. Infiltration so deep behind tonsil that it could only be coagulated and destroyed *in situ*.

Oct., 1924. Recurrence in pharyngeal scar and in neck.

March, 1925. Diathermy to growth in pharynx and diathermy puncture of neck glands.

Progress of disease very slow and painless until death in 1927. Patient edentulous and growth aseptic.

5. Male, 56. Epithelioma of soft palate and tonsil. Enlarged gland in submaxillary region.

Feb. 2, 1924. Cervical and pharyngeal operation at same seance.

May 5, 1924. Removal incomplete. Still growth in pharynx. Supraclavicular glands enlarged. Diathermy to pharynx and diathermy puncture of supraclavicular glands.

June 6, 1924. Supraclavicular glands hard and much smaller. Much pain in throat.

June 26, 1924. Diathermy to pharyngeal ulcer, and to tongue which had become involved in growth.

Sept. 2, 1924. Died.

Operated on as an eradicable case, but proved to be ineradicable. Note short duration.

6. Male, 53. Small epithelioma of right faucial pillar. No enlarged glands.

June 15, 1923. Operation on neck and pharyngeal growth.

No recurrence on Feb. 10, 1928—i. e., five years.

7. Male, 58. Epithelioma of right tonsil, anterior pillar of fauces, floor of mouth and tongue. Adherent to alveolus.

June 13, 1925. Neck operation.

June 16, 1925. Partial removal of pharyngeal growth with diathermy destruction of irremovable areas. Bone of alveolus exposed and diathermized.

Sept. 15, 1925. Bone remains bare. Wound otherwise healed.

Dec. 9, 1925. Loose sequestrum of alveolus removed. Diathermy puncture of tongue.

Dec. 16, 1927. Recurrence at cardiac end of esophagus.

Jan. 17, 1928. Died.

This was regarded as an ineradicable case. The appearance of the disease in the esophagus is noteworthy. There was no obvious local recurrence, not even in the tongue. The patient, a medical man, was able to carry on his practice until within a few weeks of his death—that is to say, for two and a half years after the first operation.

8. Female, 32. Parotid "perithelial sarcoma" of left side of soft palate extending outwards and adherent to mandible, where it could be felt externally.

Oct. 20, 1925. Enucleation tried and failed. Radium inserted into tumor.

Operation followed by sepsis of wound and general septicemia.

Dec. 1, 1925. Diathermy applied to interior of wound with the object of combating sepsis. The sepsis and septicemia disappeared and the tumor showed diminution.

Dec. 22, 1925. Second application of diathermy in the hope of destroying the tumor.

Jan. 14, 1926. Wound healed. Tumor disappeared.

No recurrence on March 5, 1928—i. e., two years and three months since last diathermy operation.

9. Male, 57. "Malignant type of mixed parotid tumor. Endothelioma or cylindroma," situated to left of center of soft palate.

Jan. 4, 1927. Removed elsewhere and radium applied.

May, 1927. Local recurrence.

Aug. 1, 1927. Neck operation; three enlarged glands "doubtfully infected." At same seance diathermy removal of entire soft palate and tonsil.

No recurrence on March 19, 1928.

10. Male, 67. Epithelioma upper part of right tonsil. Enlarged gland at angle of jaw.

Jan. 23, 1928. Neck operation, one gland epitheliomatous. Wide diathermy removal of tonsil.

A stormy convalescence with formation of communication between pharynx and neck wound. It closed later.

11. Male, 53. Epithelioma of left faucial pillar, soft palate and alveolus. Tonsil apparently not involved. Enlarged gland at angle of jaw.

Feb. 29, 1928. Neck operation. Enlarged gland proved to be epitheliomatous.

March 6, 1928. Diathermy removal of faucial pillar, left half of soft palate and left tonsil.

12. Male, 62. Epithelioma of right vocal cord.

June 23, 1927. Laryngofissure. Diathermy applied to raw surface after removal by scalpel of cord, ventricular band, etc. Convalescence stormy. Neck wound became septic.

Nov. 2, 1927. No recurrence.

REMARKS.

In the commentary on these cases, the last four will be excluded as being too recent. Of the others, eight in number, the growth could not be removed *en masse* in four. Of these, one showed imperfect removal and died in seven months; one showed apparent complete removal and local recurrence in eleven months; one showed recurrence in sixteen months, and one (perithelial sarcoma, Case 8) shows no recurrence after two years.

Of the four removed *en masse*, one shows no recurrence after five years; one no recurrence after nine years. One recurred in two and a half years, and one recurred in five years. That is to say, all four cases show freedom from cancer for from two and a half years upwards.

Of the same eight cases, seven were epithelioma, and of these, five affected the tonsil, only two being extratonsillar. In all the five tonsil cases recurrence took place, although one survived free of the disease for five years. Of the two extratonsillar cases, neither of which had infected cervical glands, neither has recurred after five years and eight years, respectively.

The numbers are small, yet they suggest that epithelioma of the tonsil, whether originating there or spreading thither from the neighborhood, is more liable to recurrence than epithelioma of the pharyngeal mucosa not affecting the tonsil. Professor Schmiegelow remarks that when the cervical glands are infected, recurrence is usual, and my cases corroborate him. Perhaps it is not fallacious to regard the tonsil as being itself a lymphatic gland covered with mucous membrane.

Taking now the eight cases as a whole and including sarcoma, five show no recurrence up to two years; three show no recurrence after three years, but of these one recurred after five years; the other two show no recurrence to date. It is evidently unwise to regard a three-year period of freedom from recurrence as equivalent to permanent cure.

This survey leaves out of count one important consideration. Even if we take the cases in which recurrence took place, we can still claim for all of them, save the one in which removal failed, a complete interval of comfortable life varying in duration from eleven months to five years.

The fact is that when the pharyngeal growth and the cervical glands are removed by a diathermy operation, the evolution of pathologic events and of symptoms is profoundly altered. Thus in two of our four recurrent cases, although the disease recurred locally, it did not progress there, and only in one of them did cervical gland infection reappear. In a third there was no local recurrence at all, but the disease suddenly appeared at the cardiac end of the esophagus. It would almost appear as if diathermy exercised some kind of local inhibitory action upon cancer. Furthermore, in all of these recurrent cases the end was quiet, gradual and free from severe pain, the septic element, which plays so powerful a part in the tragedy of throat cancer, having been permanently eliminated.

In a word, it may be said that although the cancer still persisted, diathermy had, as it were, driven it underground.

With regard to the results in ineradicable cases, every now and then, as Cases 7 and 8 show, an apparently hopeless case can be rescued, at all events for a time. On the opposite side of the balance sheet, however, we have to place that large class in which the disease is so far advanced when we first see the case that it is inadvisable to use diathermy at all. And this class is much larger than it ought to be, as we have already remarked. Every effort ought to be made, therefore, to educate the general practitioner to seek expert aid as early as possible in all forms of pharyngeal ulceration, particularly in patients over middle age.

With regard to those cases in which diathermy can be applied with a reasonable hope of relieving pain and restraining extension, we have had quite a large number of satisfactory

cases. We have obtained temporary benefit, that is to say. To expect more of any remedy for ineradicable cancer would be asking too much, perhaps, in the present state of our knowledge.

Merely as the agent of this temporary benefit, however, we can regard diathermy as one of the best palliatives in our possession at present.

Let me remind you once again that in these circumstances diathermy may be profitably associated with the use of radium and of deep X-rays.

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III.

VASOMOTOR AFFECTIONS OF THE INTERNAL EAR.

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Vasomotor disturbances may be considered as among the most pathognomonic and the most important in affections of the internal ear. These vasomotor troubles depend directly on the sympathetic system.

We have recently made a series of experiments on the action of the sympathetic in regard to the labyrinth. These experiments have been either peri-carotid sympathectomy or injections of vasomotor drugs (stimuli or inhibitors of the sympathetic; stimuli or inhibitors of the parasympathetic). Our study has dealt also with the circulatory labyrinthine troubles caused by the compression of the vertebral artery or of the "common carotid artery," or by peripheric vasodilatation (warm bath test).

These different experiments, of which the results agree with those obtained by Terracol (perivascular sympathectomy, section of the cervical sympathetic, injection of suprarenin in the superior cervical ganglion), allow of our bringing forward some conclusions on the vasomotor affections of the internal ear.

For a long time the action of the sympathetic on the labyrinthine circulation, on the one hand, and on the secretion of the endolymphatic fluid on the other, has impressed otologists. It is astonishing that the study of it has not been further developed, for in 1904 Ferreri pointed out certain affections of the internal ear in connection with some sympathetic lesions and spoke of "Glaucome," a term used again these last years by Aboulker and by myself, to designate the hypertension of the endolymphatic fluid.

Escat, on the other hand, in a very complete work published in 1906, on arteriosclerosis of the labyrinth and of the acoustic

centers, has devoted a large part to vascular spasms in the pathogeny of Ménière's disease.* But it is especially Lermoyez who (in 1919) has insisted on the vascular labyrinthine phenomena and their clinical manifestations. He showed the importance of angiospasm of the vestibular or cochlear branches of the internal auditory artery in certain cases of deafness or vertigo.

Having observed a typical angiospasm of the anterior labyrinth in a patient affected with Raynaud's syndrome, Lermoyez drew attention to the similarity of structure between the two situations, digital and labyrinthine.

The stenosis of the internal auditory artery, which is brought about slowly, causes a gradual anesthesia of the ear—that is to say, deafness. But then the spasm ceases suddenly; the blood rushes again into the labyrinth which is stunned by it. This too sudden afflux, which in the fingers produces ordinary pain, causes here both the special pain of the cochlear organ, which is the buzzing, and the suffering of the vestibular organ which is the vertigo. Besides that, it abolishes the anesthesia of the ear—i. e., deafness.

And thus Lermoyez has summed up under the name of "the vertigo that makes one hear" a particular labyrinthine affection, which seems to be connected really with a spasm of the internal auditory artery.

It is expedient, from another reason, to oppose this syndrome to Ménière's disease: this sudden paroxysmic, apoplectic and recurrent vertigo, which breaks out in the repose of full health and which was so long united with cerebral apoplectic congestion.

In Ménière's disease, and in Lermoyez's syndrome, the phenomena are the same, but they make their appearance in reverse order.

The vertigo which in one case inaugurates the fit, in the other ends it. In one case it suddenly impairs the health, in the other it restores it; in one case it brings deafness, in the other it cures it; but in both affections it always comes unexpectedly.

A table for comparison will better explain the opposition of the two syndromes:

Ménière's Disease	*Lermoyez's Syndrome*
Good hearing	Good hearing
Sudden vertigo	Sudden vertigo
Sudden deafness and decreasing	Gradual deafness
Slow recovery	Quick return of the hearing

The angiospasm would very well explain the onset of vertigo and the disappearance of the deafness. These two symptoms being dependent upon the same cause: the removal of the barrier of the labyrinthine artery. These are simultaneous phenomena: hearing does not return because the vertigo sets in, but when the vertigo sets in, this vertigo is not the cause of the return of hearing; it is only the disagreeable witness to its presence.

If one has put in perfect opposition, from a clinical point of view, Ménière's and Lermoyez's vertigo, it has been the same as to pathogeny. One wished to oppose the congestion and even the labyrinthine overflow of the first, to the ischemia of the second, consecutive to the arterial spasm.

Nothing is more debatable, however, than the pathogeny of Ménière's disease: hypotheses most contradictory and most opposite have been turn by turn in vogue. At the present time it is still an obscure question which does not appear to be near solution.

The first in date of these hypotheses was that of Ménière himself, viz., that the affection was caused by an intralabyrinthine hemorrhage: this, he said, had been proved at the necropsy of a young girl who had died after several severe attacks.

Alexander, however, strongly attacks this opinion and advances the hypothesis that it is rather a question of a patient suffering from leukemia with leukemic hemorrhage in the labyrinth.

Quincke, on his side, has stated his opinion that Ménière's disease is due to a phenomenon similar to that which causes the "dermic circumscribed edema." He thinks, no doubt, of an acute angioneurotic edema in the labyrinth, followed by an endolymphatic hypertension.

According to Wittmaack, it is a question of intermittent variation of hypertension of the endolymphatic fluid, entailing

modifications of the intralabyrinthine pressure. This author, relying on his experiments, thinks that the hypertension is due to a hyperplastic catarrhal state of the middle ear, with diffusion of toxic substances through the "windows."

Escat postulated a different pathogeny for each clinical form of Ménière's disease.

(a) The paroxysmic form, with remission absolutely complete, seems to realize the intermittent labyrinthine claudication by spasms of the internal auditory artery. The mechanism of the giddy fit would be then the same as that of Lermoyez's syndrome.

(b) The apoplectic form, in which the three symptoms reach their apogée from the first "ictus." This postulates the sudden, complete and irremediable loss of hearing. This form has incontestably as its most constant substratum an intralabyrinthine hemorrhage.

The rupture of the internal auditory artery is really here the equivalent of the rupture of the artery in cerebral hemorrhage and of epistaxis.

The Danish authors, S. H. Mygind and Hwidt, are of the opinion that the fits of vertigo are in connection with an intermittent increase of the endolymphatic pressure, but do not give any indication of the cause of this hypertension.

Other authors, as Bárány, Aboulker, Quix, have thought rather of an intracranial origin. They see in a hypertension in the area of the lateral cisterna, causing a pressure on the eighth pair, and also on the medulla oblongata, the pons and the cerebellum, the cause of Ménière's vertigo.

Then Quix asserts that this hyper-pressure must act above all on the otolithic apparatus by the way of the "ductus endolymphaticus."

S. H. Mygind makes a similar suggestion, while Thornval, who thinks also of an affection of the otolithic system, places the original lesion in the medulla oblongata.

Kobrak ascribes the pathogeny of Ménière's vertigo to angioneurotic fits of the eighth pair of cranial nerves, depending on two principal causes: (1) a great instability of the vago-sympathetic system, and (2) a local labyrinthine trouble.

By experiments with pharmacodynamic substances (atropin, pilocarpin, adrenalin) Kobrak tries to find in each individual

case whether it is a question of vagotony or one of sympathicotony.

The examination of these different theories shows the important place given to vasomotor troubles in the pathogeny of Ménière's disease.

Whether it be Quincke, Wittmaack, S. H. Mygind, Hwidt, for whom it is a question of an endolymphatic hypertension, which can but be closely connected with vascular phenomena, or Kobrak, who considers the direct cause in the vagosympathetic system, the vasomotor disorders—vasoconstrictor or vasodilator—seem really to constitute a pathogenic primordial element.

And Vernet, taking as two extremes Lermoyez's ischemic-vertigo and Ménière's vasodilatation vertigo, thinks that between these two syndromes there is place for a whole series of troubles ranging from ischemia to congestion.

If with Lermoyez's syndrome there may be a spasm of the internal auditory artery or one of its branches—vestibular or cochlear artery—there may, on the contrary, exist a vasomotor stasis in the territory of this artery or one of its branches.

This congestion will expel the access, as the latter is expelled by the sanguinous irruption which comes to destroy the labyrinthine anesthesia on Lermoyez's patients.

The vasomotor stasis of the vestibular artery may cause a giddy sensation apart from any other manifestation. All this agrees with the opinion that vertigo is essentially a phenomenon of irritation.

All otologists know that vertigo disappears with the destruction of the labyrinth and that an irritation of the internal membranous ear or of the vestibular nerve is necessary to cause the giddy sensation.

One sees, on the other hand, the reflex vasodilatation which locally accompanies the least irritation of the tympanum and which, when this irritation is too violent, also produces vertigo.

A strident noise, irritating the original filaments of the cochlear nerve, may also produce vertigo by reflex vasodilatation.

These few examples, joined to the experiment, show well the connection which exists between the vertigo and the phenomena of vasodilatation in the area of the labyrinth.

By what inward mechanism is this irritation of the vestibular nerve produced? Is it simply a disturbance of the vasotonic equilibrium of the capillaries, under the more frequent form of stasis, which acts directly; or is there, as a necessary medium, some modifications of the endolymphatic pressure?

It is not possible to give an explanation of this problem in the present state of our knowledge.

One cannot in all cases state with precision the connection between the capillary circulation and the endolymphatic pressure. Perhaps one might see in this increase of the endolymphatic pressure a disorder in relation with capillary stasis. It is the same as in edema in peripheral stasis; and it is that idea which has guided the surgical treatment of certain cases of vertigo: either one acts by decompressive trepanning, or one performs my operation, viz., the opening of the saccus endolymphaticus. On the contrary, is it possible, as Vernet thinks, to consider these cases of endolymphatic hypertension and vertigo at the same time as the sympathetic phenomena associated?

It is difficult to reply in a decided manner, but it is nevertheless true that, whatever the inward mechanism of production of these vertigoes, it seems to be, in Ménière's disease, closely connected with the vasomotor phenomena, and hence with the sympathetic equilibrium.

From this rapid review of the different affections of the labyrinth in which one has brought in, from a pathogenic point of view, the probable existence of vasomotor troubles, one must separate some precise ideas:

Vertigo, when a sign of vestibular suffering, seems to be caused by reverse vasomotor troubles; for example, it is produced by the sudden vasodilatation which follows the spasm in the syndrome of Lermoyez.

The vertigo which, on the contrary, accompanies a rather pronounced ischemia of the labyrinth, disappears after sympathicectomy, of which the consequence is a vasodilatation of the labyrinth.

Tinnitus means cochlear suffering and follows apparently the same rules, being produced either by vasoconstriction or by vasodilatation; it may be cured or ameliorated, according to the case, when one provokes in the region of the labyrinth the vascular action contrary to that which has caused it.

Vestibular reflectivity seems to be precisely proportioned to the vasomotor modifications. To vasodilatation corresponds hypoexcitability—to vasoconstriction, hyperexcitability. All experimental tests and all the clinical observations agree on this subject, and this is perhaps the most important and the surest result obtained up to the present in the study of auricular spasms.

Deafness appears to accompany ischemia of the anterior labyrinth, caused by vasoconstriction. Sometimes it may be ameliorated or disappear by the action of the vasomotor phenomenon, contrary to that which caused it, that is to say, the vasodilatation. But if we bear in mind the tenderness of the cochlear sensorial elements we shall understand that a prolonged vascular obstruction may involve this irremediable damage and definitive deafness.

Is it possible to select from these scattered observations a clinical syndrome of the vascular spasms of the internal ear? I think that we may now settle the chief symptoms, reserving the right to modify them in detail when research on the vasomotor mechanism of the labyrinth is more advanced. The arterial spasm provokes in a determined labyrinthine territory, ischemia, more or less pronounced, which entails signs of suffering of the sensorial apparatus, namely, vertigo and buzzing, functional troubles and vestibular hyper-reflectivity. The term "spasm" implies a close participation of the periarterial sympathetic in connection, in most cases, with the loss of neurovegetative equilibrium.

The angiospasmodic syndrome of the labyrinth will consequently include: (1) Tinnitus; (2) deafness, vestibular hyperexcitability; (3) sympathetic hypertony. These symptoms have always been in accord in the various investigations which I have made, and constitute a constant syndrome with certain modifications, due to the particular ground submitted to the vasoconstriction: anterior labyrinth, posterior labyrinth, or both together.

Besides this syndrome of arterial resistance or hypertonicity of the labyrinth, we must place the syndrome of hypotony or laxity, in which we shall meet the classical signs of the sensorial suffering, but with vestibular hypoexcitability and sympathetic hypotony.

These two syndromes may, moreover, succeed each other alternatively in the same patient.

According to the individual, one observes, in fact, different reactions of the vegetative system under the influence of various causes.

But most often one meets with a hypertonic or parasympathetic syndrome, which, as we have seen, permits us to classify individuals into vagotonic and sympathicotonic. One meets in clinics, fairly often, subjects with whom the disequilibrium seems to be caused by a global hyperexcitability of the vegetative nervous system. It is this particular state that authors have described under the different names of "neurotony" (Guillaume); "total disequilibrium of the whole system" (Laignel-Lavastine); "vegetative dystony" (Sicard); "amphotony" (Danielopolu).

However, this vegetative dystony may be in some patients with vagotonic predominance, in others with sympathicotonic predominance, and with certain subjects, as Harvier and Chambrun have pointed out, hypertony predominatees over the sympathetic on the level of one organ, and the parasympathetic on the level of another organ.

The result is a truly perplexing instability of visceral innervation.

The most diverse causes will be found as the origin of the vagosympathetic troubles and hence of the labyrinthine vascular spasms. The causes may be either mechanical, endocranial, toxic or even simply psychic.

The most important causes susceptible of acting on this regulating apparatus are undoubtedly the action of the nervous system and the action of the endocrine glands, above all, the hormone of the suprarenal gland—adrenalin. Is the hypertonicity of the arterial coat during the spasm due to the hyperactivity of the sympathetic or to a hyperfunction of the suprarenal?

These are, as Dumas has pointed out, questions to which it is impossible to reply in the present state of our knowledge, but it is certain that it is in this direction that we must continue our researches.

IV.

HEADACHE FROM THE STANDPOINT OF THE RHINOLOGIST.

By Henry Dintenfass, M. D.,

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Of headaches from the rhinologic standpoint there would seem to be little to say that has not already been said. However, there are some angles and some aspects that do not get into the current literature. One cannot have had intimate contact with this subject without realizing that the accepted views, particularly as regards diagnosis and treatment, are constantly changing.

For example, a little over a decade ago there was distinct skepticism as to the part assumed by the nose in the production of facial and head pain. Later the medical profession came to recognize that intranasal and accessory sinus diseases were in many cases the cause of obscure headache and neuralgia, which have been classed as migraine, hemicrania and similar affections. Today this knowledge has even spread to the laity. It is nothing unusual, for instance, for a patient to speak with complete assurance of a sinus headache, sometimes even mentioning the particular sinus and demanding relief. The result has been that the rhinologist, in his effort to satisfy the patient, has often been forced to adopt radical therapeutic measures unnecessarily. Only recently a patient came to me with a self-made diagnosis of a sinus condition and suggested that her right antrum be punctured because of severe, agonizing pain over the right cheek and forehead. My examination showed that the sinuses were absolutely negative, and when I explained that the teeth might be the cause of the trouble she stated that an X-ray had already been taken and that her teeth were perfectly sound. I insisted on further X-ray studies, however, and, much to her surprise, two impacted molars in her upper jaw were discovered, the removal of which cured her headache. It was evident that treatment directed to the sinuses would not only have been unjustifiable but would have in-

creased her discomfort. Many times, too, have we seen patients with definite abnormal and diseased conditions in the nose and advised an operation, predicting that when this were done they would be for all time relieved of their pain. After the operation it was found that even with an excellent postoperative result headaches were not only as severe as before but new symptoms were added to the old.

So that we can best understand the reason for these shortcomings let us enumerate some of the varying anatomic and pathologic conditions met with in this region.

1. The nose as the most protruding portion of the face is easily exposed to injury, resulting in deflected septa and other abnormalities. In 75 per cent of the adults in the Caucasian race there is deflection of the septum, and in nearly a quarter of these the deviation is sufficiently pronounced to cause symptoms.

2. In the human being the nose has two sinuses which have no gravity drain, the maxillary and sphenoidal. The disadvantage here is obvious, since infection in these sinuses with suppuration is less apt to subside spontaneously than in the frontal sinus, where the natural drain is favored by gravity. For this reason in the animal that spends much of its life with its nose pointing downward or to the earth, the maxillary and sphenoidal sinuses are never involved by disease.

Incidentally, this would seem to support Darwin's theory concerning man's descent from the animal. Nature had apparently intended that the sinuses be well taken care of by placing the ostia at the bottom of their respective cavities. In the course of evolution, however, when the erect posture was assumed, the location of the orifices, with the drawing upward of the head, were shifted to their present site, where they no longer serve for natural drainage.

3. The upper air passage from birth to death is subjected to recurrent inflammatory attacks, often leaving an indelible imprint on the tissues. These range from the simple ordinary colds to the severe streptococcic infections.

4. The center of sensory nerve distribution of the nasal cavity is Meckel's ganglion. This bit of nerve tissue is about 5 mm. in length and is situated in the sphenomaxillary fossæ

behind and above the posterior tip of the middle turbinate, lying just beneath the mucous membrane. It is the second largest nerve center outside of the cranial cavity and, according to Sluder, it innervates and is intimately connected with every region treated by the otorhinolaryngologist. Its distribution is so extensive as to give symptoms directly and reflexly to practically every part of the body. It receives its sensory roots from the superior maxillary branch of the trifacial division of the fifth nerve and so is directly associated with that important nerve structure, the Gasserian ganglion. It is in close relationship to the sphenoid sinus, the posterior ethmoid cells and the maxillary sinus. It is apparent, therefore, that in all questions pertaining to head pains Meckel's ganglion assumes a prominent rôle.

The study of nasal headaches then resolves itself into a consideration of not only the abnormal conditions of the nasal cavities but also affections of the adjacent nerve structures.

The actual pathologic factor which gives rise to the subjective sensation of pain is some type of trauma. In the case of the sinuses, the trauma takes the form of pressure. In the nerve structures it acts as an irritation. Pressure may result in several ways. One, in which the lining mucosa of the sinus becomes so congested and swollen as to impinge on the lining mucosa of the opposite side, thus meeting and obliterating the sinus cavity. Another, when the outlet of the sinus becomes more or less occluded, blocking the drainage of secretions with resultant pressure on the already swollen and inflamed mucosa. A third, where the pressure is negative in character and the so-called vacuum headache is produced. This usually involves the frontal sinus and is supposed to be due to the closing of its normal aperture and absorption of the contained oxygen. It is accompanied by tenderness at the upper and inner part of the orbit at the attachment of the pulley of the superior oblique muscle. (Ewing's sign.) Many observers believe that headache of this character is present when relief occurs following inflation of the nose after a previous shrinking of tissue at the frontal sinus opening. My own personal experience with this type is that very few of these cases really exist. Pressure may also be responsible for headache in other ways than by occlusion of a sinus. I have reference to contact pressure produced

by close apposition of the turbinate bones to the septum. This is apt to occur if the turbinates are congested from pollen and other allergic sources or if growths like polypi are present, and is particularly well pronounced when the septum is deflected or has a spur or ridge upon it. An example of headache due to contact pressure that was quite unusual was in a case where the turbinates were congested from an allergic condition. The patient, a woman, complained of severe headache at 6 o'clock every evening, accompanied with sneezing and running of the nose. She had been treated for sinus trouble, including antrum punctures and irrigation, with no appreciable improvement. Careful questioning concerning the history elicited the fact that these attacks were coincident with the arrival of her husband from his daily duties. He was employed in a riding academy and his working garments were a distinct source of allergy to the patient. The mere expedient of changing his apparel before entering the house was sufficient to prevent further headaches in his wife.

Under the classification of headaches from trauma due to irritation belong the true nasal neuralgias. This includes abnormalities of Meckel's ganglion and other adjacent and connecting nerve structures and is probably caused by a certain toxicity liberated by the infecting microorganisms. Dr. Greenfield Sluder was the first to establish the importance of diseases of Meckel's ganglion as a distinct entity. He said that affections of this organ were not only responsible for pains that included every region in the head but that a sympathetic syndrome simulating hyperesthetic rhinitis or hay fever was sometimes associated. He proved that this was a true neurosis or nerve irritation by the temporary relief of symptoms immediately after the application of a 10 per cent cocain solution over the ganglion area. Recently Ruskin and others have gone a step further, maintaining that Meckel's ganglion affections can cause bizarre manifestations far removed from the nose, such as pain in the neck and shoulder, pain in the back, intercostal neuralgia and even gastric pains, diarrhea and hiccoughs and that the cocain application to the ganglion region has a distinct beneficial effect on these conditions. Not so long ago a patient in the Graduate Hospital developed hiccoughs one week after a gall bladder operation. The usual remedies were

used for several days with no effect. I suggested that cocain to Meckel's ganglion be tried and was agreeably surprised at the result, the hiccoughs subsiding immediately.

In the consideration of the diagnosis and the symptomatology it may be said that headaches due to intranasal lesions occur daily at about the same time or under the same conditions. Headaches from sinus disease show certain characteristics. For example, there is the tendency to remission, either partially or completely, during the day. This is the result of better drainage from change of posture, with the subsequent evacuation of sinus contents. Then there is the intolerance to alcohol and tobacco. Their use increases the congestion in the head, adding to the already swollen and inflamed mucosa. The relief afforded sinus patients when hot towels are applied to the face is also important from the diagnostic viewpoint. This is brought about by the stimulation of ciliary motion, favoring the drainage from the sinuses by enlarging the ostia. Considering the sinuses more specifically, we find that a peculiarity of frontal sinus headache is that it is usually present on rising in the beginning of the day, lasts for several hours and either remits partially or completely in the afternoon. The location of the pain here is at the upper inner corner of the orbit or over the eyebrow. Since this is one of the sinuses where drainage is favored by gravity, it follows that in the early morning hours, after the patient has been on his back for some time, the discomfort is greater than at any other period. In headaches from affections of the ethmoid sinus, especially in children, swelling and edema are sometimes seen, strange to say, over the frontal area. This is due to the extension of the deeper lymphatic and venous congestion to the skin surface and is not to be mistaken for a frontal sinus affection. Ross Hall Skillern points out that the frontal sinus in patients under six years of age is absent or very small in size (no larger than a pea). Therefore, swelling in this region could only come from ethmoid involvement. This is of extreme importance when radical procedures are to be considered.

When the anterior ethmoid cells are involved the pain may be frontal or felt as a dull pressure between the eyes. In the posterior ethmoid and sphenoid affections, the pain is usually experienced as a diffuse sensation in the occiput but may ex-

tend into the mastoid and temporal regions. Because of the close proximity of these sinuses to the sphenopalatine ganglion the complexity of the pain varies, and ganglionic syndrome manifestations, such as pain in the back and shoulder, etc., may also be present. In pain from antral inflammations the symptoms may consist of a fullness in the malar region, with a disagreeable sensation in the alveolar processes of the teeth, or, as in a case that came under my observation, may be referred to the frontal region on the same side. This patient complained so severely of pain over the left forehead and frontal area that the frontal sinus was thought to be involved. Transillumination and subsequent puncture showed that the maxillary antrum was entirely at fault. Since the antrum opening is located almost at the top of the cavity the degree of headache is decidedly lessened when the patient is in the prone position. Lying on the healthy side also aids in the drainage and relief of symptoms. That antrum disease may also be responsible for Meckel's ganglion syndrome has been well shown by Alden. It is not common, however, because the pathology of maxillary sinus conditions is usually limited to the floor and nasal wall of the sinus and not to the upper part where it is adjacent to the ganglion.

With reference to the order of frequency in the production of headache, Mosher believes that the frontal sinus should be considered first, then the ethmoid and lastly the maxillary. Regarding the complete absence of pain in attacks of sinusitis, Skillern is rather skeptical. He states that he has never met a case in his own practice in which either tenderness or pain had not been present.

In the differential diagnosis of intranasal headaches from the socalled migraine headaches it should be remembered that in the latter there is a periodicity of occurrence with wide intervals of freedom. In migraine the commencement occurs early in life and is hereditary in nature and is usually associated with vomiting and similar phenomena representing auræ. Mithoefer calls our attention to a unilateral headache, which sometimes is diagnosed as migraine when nasal examination does not disclose pathology. He believes that there is an anatomic variation on the floor of the frontal sinus which narrows the ostium so that complete closure takes place as a result of a

mild hyperemic state of the nasal mucosa following some systemic derangement.

A distinction must also be made between Meckel's ganglion syndrome and Gasserian ganglion affections. In the former the pain is much more severe at night and not limited to the trifacial tracts. There is also no marked peripheral sensitiveness as that observed in trifacial neuralgia. Barnhill states that affections of Meckel's ganglion result in minor neuralgias of the head, while that of the Gasserian ganglion produce the major neuralgias.

The possibility of mistaking an intracranial lesion for an intranasal one is also worthy of consideration. Cushing reminds us that one would hardly expect to relieve headache from the former source by surgical procedures in the nose, yet it is a well known fact that not a few patients with brain tumor have been operated upon for supposed sinus conditions.

Pain from impacted teeth has been mistaken for sinus pain, reference to which has already been made. Instances of this kind are not uncommon.

Another condition which may cause confusion in diagnosis is pituitary disease, as the following case will illustrate. Several years ago Mr. H. L., 28 years of age, came to me for relief of a splitting headache, which had existed for a number of months, and was gradually increasing in severity. The examination disclosed that practically all the sinuses in the head had been operated upon. The patient was very much depressed and stated that if something were not done for him soon he would commit suicide. It was evident that the etiology of the pain was not of sinus origin. On looking for a possible cause of the trouble it was noticed that the patient's face and head were unusually large and that his hands were also of extreme size. The possibility of pituitary disease suggested itself. An eye examination disclosed the characteristic slicing down of the form fields above on the temporal side and confirmed the diagnosis of pituitary trouble. Dr. Cushing of Boston operated upon him for this condition with the most gratifying results. He was relieved of his headaches immediately and has been free from pain ever since.

In establishing and confirming the diagnosis of sinus disease for the cause of headaches shrinkage of the mucous membrane

by cocain with the help of adrenalin and ephedrin is necessary so that a better view is possible. If the headache is completely relieved by the shrinkage alone, it may be concluded that the headache is the result of mechanical factors. The fact that pain was not relieved by shrinkage does not rule out pressure, for occasionally the anatomic configuration is such that it is impossible to place the shrinkage solution where it will do the most good. The middle turbinate bones should be carefully examined and cotton should be left in the middle meatus long enough to determine the presence of an outflow of pus, if at first not apparent. Anterior location of pus is usually frontal in origin, but may also involve the anterior ethmoids. Pus in the posterior half of the middle meatus may be posterior ethmoidal, antral or occasionally sphenoidal. The location of the pain should receive careful consideration. In the case of the sphenoids evidences of a lateral pharyngitis might be the only suggestion for a proper diagnosis. Transillumination, X-ray and sinus puncture wherever possible are of value. Lipiodol injections, according to the method of Proetz, have been a great diagnostic step forward and will show the presence of thickened mucosa and mucoceles in sinuses which hitherto were demonstrable only upon the operating table.

Regarding antral irrigation, a word of caution might be appropriate. Mithoefer believes that washing out the antrum cavity in acute maxillary sinusitis is not only painful but could easily be responsible for mixed infections and septic conditions sometimes resulting in death. He states that the temperature should be taken before irrigation and that if fever exceeding 100° F. is present lavage should not be attempted. This coincides with the experience of Dr. Walter Roberts. In acute antral cases, where urgent therapeutic measures are necessary, the writer has been in the habit of puncturing the maxillary sinus without washing out the cavity. The mere insertion of the needle associated with the placing of the patient on his sound side have been sufficient to produce relief.

No symposium on nasal headaches would be complete without mentioning the value of ephedrin. In the entire domain of otolaryngology there is not a single drug that can be compared with it. It has most of the advantages of adrenalin and cocain

without their disadvantages. It is useful both in the diagnosis and treatment of nasal conditions. Its property of enlarging the sinus ostia without the secondary congestion that usually follows the other drugs has been the means of not only obviating the necessity of sinus punctures but also of shortening the duration of sinus affections. It is most effective when used as a spray in a 1 per cent aqueous solution.

Due credit should also be given cocain. Sluder proved the efficacy of this drug as a diagnostic measure in the so-called Sluder's or Meckel's ganglion syndrome by the remarkable relief following an application of this alkaloid to the sphenopalatine area. I can recall many instances in my own practice where this was done with excellent results. This procedure is easy of accomplishment if the cotton-tipped probe is placed upward and backward into the nose as far as it will go in a direction that will exactly bisect the inferior turbinate bone. Cocain is also of distinct value, although not to the degree of ephedrin, in the determination of sinus conditions. Its action here is twofold, first, as a local anesthetic from paralysis of the sensory nerve endings, and second as an astringent due to the stimulation of the constrictor fibers in the muscle walls of the arterioles. It should be emphasized, however, that if a headache is relieved by cocain application to an appropriate part of the nasal mucosa the cause of the pain may not always be of nasal origin. This is well exemplified in the relief of premenstrual headache by a prolonged cocain block to the sphenopalatine area.

In conclusion, it is evident that as our knowledge concerning the various types of headaches is increasing it is easier to differentiate the causative factors involved. It follows, therefore, that before intranasal operative interference is resorted to for the relief of headache the surgeon should be reasonably certain that the patient will be benefited by the operation. No more common abuse exists in rhinologic work than these operations upon the nose undertaken to relieve headaches which have nothing to do with nasal conditions. A careful, systematic, painstaking attempt to establish a definite diagnosis is absolutely essential before any surgical procedure is suggested.

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V.

DIAGNOSIS OF ANTRAL INFECTION.*

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The requisites for the preservation of healthy sinuses are ample drainage and proper aeration. The most that the sinusologists can hope for in the treatment of early sinus disease is to accomplish Nature's purpose by the simplest possible method, and that is to establish ventilation and drainage.

The maxillary sinus is unique in its anatomic make-up, because of the fact that the natural ostium is at the uppermost level of the sinus. Bearing in mind this anatomic anomaly in its relationship to drainage, one must add the lateral nasal wall, the ethmoid infundibulum, the hypertrophied middle turbinate and the deflected septum.

The antrum is anatomically peculiarly situated. It is apt to become infected from below by the teeth through actual pathology, by continuity and contiguity of infected teeth, or through surgical intervention during extraction or treatment of the teeth. From above, the ostium of the antrum is in the pathway of the overlying sinuses. During the course of infection of the other sinuses the pus is apt to drain into the maxillary antrum and convert it into a cesspool.

The antrum, being the largest sinus of the nasal group of sinuses, is susceptible to its own inherent infections, exclusive of the aforementioned facts. Very often we find multiple ostia, diverticula, septa, which may be partial or complete, often producing double maxillary sinusitis. To reiterate, the ostium is at the uppermost level of the sinus; therefore, drainage is readily impeded. Ross H. Skillern, J. Parsons Schaeffer and Harry Neivert have called our attention to the fact that there are often multiple ostia and that instead of the ostia open-

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ing into the ethmoid infundibulum, they often open directly into the lateral nasal wall.

The overdeveloped maxillary sinus plays an important part in its relationship to the dental plexus, due to the thinness of the bone and the close proximity of the dental structures to the floor of the maxillary antra. It is in this type of case that dental devitalization may take place when chronic maxillary sinusitis occurs, and often vice versa.

It is imporant to describe the manner in which we observe the infections of the maxillary antra, bearing in mind that we base our diagnosis upon the correlation of the symptoms and signs of the individual accessory sinuses.

1. We eliminate all possible nasal infections outside of the antra. This is accomplished by shrinkage, suction, packing and posture, so as to prevent the pus from other sinuses from draining into the antra while under observation.

2. Transillumination.—We transilluminate to determine whether we have clarity or opacity. (We must remember that it has some value for a confirmative diagnosis, but it is at no time worthy of replacing the more reliable endonasal diagnostic procedure.)

3. Our next step is puncture of the antra. The findings are significant and often conclusive. When pus is found of the granular type, we know that it is significant of chronic sinus infection. When the antrum is punctured and no pus is found, we assume that the antrum emptied itself through the ostium, or, for the time being, is free from pus, or that the sinus is normal.

4. Radiographic studies are an important and valuable addition in our aids to diagnosis—in fact, sometimes indispensable.

5. Lipiodol Injections.—Lipiodol is one of the radiopaque substances, which, when employed properly, will definitely visualize gross pathology in the antrum, especially when X-rays are taken repeatedly. It will give us an inkling as to the functional capacity of the ciliated epithelium in eliminating the oil from the antrum, and the time consumed before the antrum is completely emptied. Lipiodol, being a liquid, will,

when forced into the antrum, seek its level, and will only occupy the tissue interspaces. Lipiodol has no therapeutic value in maxillary treatment.

6. Cytologic Studies.—Smears are made from the pus found in the antrum during antrum puncture, and studies are made to differentiate the type of cells that predominate. In the acute we find the polymorphonuclear; in the chronic, the lymphocyte; in the subacute, the polymorphonuclear. Should there be a tendency to chronicity, then the lymphocyte prevails.

To delve into the subject of antral pathology, I have thought it best to simplify the subject by arranging the following clinical classification:

- Antral Infection
  - 1. Suppurative
    - (a) acute
    - (b) subacute
    - (c) cesspool
    - (d) chronic
      - hypertrophic
      - atrophic
      - mixed — hypertrophic and atrophic
      - polyps
      - cysts — infected and not infected
  - 2. Nonsuppurative
    - (a) subacute maxillary sinusitis undergoing resolution
    - (b) hypertrophic
    - (c) degenerative and retrograde type — (1) atrophic; (2) polyps
  - 3. Cysts
    - (a) Dental
      - (1) root or radicular
      - (2) follicular or dentigerous
      - (3) multilocular
    - (b) Maxillary
      - (1) mucoid
      - (2) hydrops
      - (3) polypi undergoing cystic degeneration

Having obtained a history, and clinical and endonasal examinations having been made, let us assume that our diagnosis was suppurative maxillary sinusitis.

In the suppurative group we are confronted with four types, namely, acute, subacute, cesspool and chronic. They all present the same clinical phenomena—respiratory disturbance, free pus and local symptoms characteristic of maxillary infection. Assuming that there was darkness on transillumination and pus was found on puncture of the antra, how then are we to differentiate these four groups? Transillumination will disclose opacity and X-ray will reveal a shadow in each. After puncture and irrigation of the antrum in the acute, subacute and cesspool cases, we find a difference in the shading on transillumination and a change in the X-ray shadow. This is due to the fact that the intramaxillary tissues have not undergone any extensive changes and the space left, due to the removal of the pus, is readily noted. However, we must reserve our opinion in the subacute type, especially where the antrum has passed through a stormy infection, as we have found that it takes between five and six weeks for complete resolution to take place. Therefore darkness on transillumination and a shadow on the X-ray may be persistent. Nevertheless, in the subacute type, the subsequent X-rays following irrigations will show the shadow changes in proportion to the resolution taking place.

The chronic suppurative antra are divided into the following: hypertrophic, atrophic, mixed, consisting of hypertrophic and atrophic, polyps and cysts. In the chronic suppurative antra, where tissue changes have taken place, whether they be hyperplastic or metaplastic, the opacity on transillumination remains unchanged and the shadow cast on X-ray is permanent because of the tissue changes.

Lipiodol employed in suppurative maxillary sinusitis, although therapeutically valueless, is almost indispensable from a diagnostic standpoint and will aid us in revealing (a) the topography of the antrum, (b) size and shape of the antrum, (c) thickness of the mucous membrane, (d) tissue displacements which may be hypertrophied, polyps, or granulation tissue, (e) duration of the oil retention.

In the acute, subacute and cesspool cases the antrum should be completely filled with the oil, due to the fact that the gross

tissue changes are not very marked. It is also noted that the oil should be thrown off within 24 to 48 hours. In the chronic suppurative type, where tissue changes have taken place and where the ciliated epithelium has undergone change, the oil will will occupy only tissue interspaces and may remain in the antra indefinitely because of the destruction of the ciliated epithelium. Any possible congestion or tissue hypertrophies around the ostium may also prevent the escape of oil. The only means by which this type of antrum empties itself is by the mechanical method, and that is by forceful blowing of the nose and posture, rather than by the physiologic route, which is accomplished by ciliary motility. (Unfortunately, there is an economical disadvantage when employing this method of diagnosis, for all this work must be carried on in conjunction with a roentgenologist, as repeated X-ray studies are made so as to follow each step and interpret the findings as they present themselves.)

The following case will illustrate bilateral antral infection of the suppurative variety:

Right side, cesspool; left side, chronic suppurative maxillary. Case 17, X-ray 1388, No. 2577.

R. G., female, 22. Social Service worker. Headache, cough, profuse nasal discharge, eye disturbance, anemia. Duration, three years.

Antrum previous operation, none.

Other nasal operations: submucous; bilateral middle turbinectomy anterior ends; tonsillectomy.

Local symptoms: Profuse nasal discharge, bilateral; both anterior ends of middle turbinates absent. Postnasal discharge; constant stuffiness of nose.

General symptoms: Anemia, headache, eye disturbance, cough.

Pus in nose coming from middle meatus; nasal septum in good condition. Posterior ends of middle turbinates intact; operation by M. S. E.

Posterior ends of middle turbinates intact. No drainage. Right side drainage in posterior group.

Transillumination: Both sides cloudy.

Exploratory puncture: Pus on both sides.

X-ray studies: September 21, 1926. Both maxillaries cloudy. A month following did a partial middle turbinectomy anterior end on right side.

Findings in puncture following X-ray: Right side looks clear.

X-ray study following washing of antrum: Right side is improved. No change on left side; still cloudy. Oil placed in both antra. (1) Right side: oil fills antrum completely. (2) Left side: only partially filled.

X-ray study of retention of lipiodol: Some oil retained on left side.

Operation: Caldwell-Luc on left side.

Operative findings: Polyps, thickened mucous membrane and pus found.

Bacteriology: Nonhemolytic streptococcus and staphylococcus albus.

Pathology microscopic: Myxomatous tissue and areas showing a great deal of round cell infiltration, hemorrhage infiltration, and granulation tissue.

Deductions: 1. Cloud in right maxillary due to cesspool from ethmoidal discharge on right side. This was proven after the maxillary was washed several times and the right maxillary became clear. When washing was stopped for a while it again became cloudy due to collection of pus from the ethmoids.

2. Anterior ends of right middle turbinates removed, clearing up the ethmoid infection. Thereafter, no pus drained into the antrum.

3. If the cloud in the right antrum was due to maxillary infection and pathologic tissue changes, it would never clear up with irrigation and it would not have cleared up following turbinectomy.

4. Left side did not improve with irrigations.

5. The cloud still remained.

6. The lipiodol filled the cavity on the right side completely, but on the left side it was only partially filled with oil.

The maxillary antrum acted as cesspool on the right side. The left side did not improve because it was filled with pathologic tissue.

The nonsuppurative maxillary sinus is divided into the following: (a) Subacute maxillary undergoing resolution, (b) hypertrophic, (c) degenerative and retrograde types, under which belong the atrophic and polyps. In this group we have patients who have had focal infection without actual nasal symptoms. We found that in a group of 150 cases analyzed, 25 per cent suffered from focal infection and at no time were their symptoms referable to the nose. We can readily comprehend the difficulties encountered when upon nasal inspection there was no pus found and no other symptoms could be referred to the nose. In order to clarify some diagnostic points we must mention some of the outstanding observations noted in these types of cases.

1. Transillumination in the subacute, hypertrophic, granulation tissue and polyp cases will invariably give us opacity variations.

2. Negative antrum punctures should not deter us from seeking further evidence as to what extent the maxillary is involved.

3. X-ray will reveal various shadows and they must be interpreted as to their significance.

It is at this point that we must definitely rule out the resolution type of maxillary antrum secondary to infection. This is accomplished by repeated irrigations and ventilations and periodic radiographs, observing, of course, the shadow changes. In this type of case, the shadow will disappear in proportion to the resolution taking place. The antrum, filled with hypertrophic tissue, polyps and granulation tissue, will not reveal any change in the opacities on transillumination, nor will any change in the shadow be noted on X-ray, irrespective of the number of times and the frequency of the washings. In fact, the shadows and the opacities often become exaggerated after repeated irrigations, probably due to the edema of the cells caused by the irrigating solutions. Then, it is here that lipiodol is almost indispensable and should be employed, as it will visualize the intramaxillary tissue and the gross pathologic structures within the antrum. In the subacute type the oil will completely fill the cavity and may show some thickening of the tissue, while in the hypertrophic type there is a distinct tissue thickening and oil displacement. Of course, the maxillary antrum that contains granulation tissue, polyps and cysts will show distinct oil displacement, as oil can be instilled only in the spaces remaining that are not filled with pathologic tissue.

The following case illustrates a hypertrophic type of nonsuppurative maxillary sinusitis:

Case 5. X-ray 1563.

B. M., female, 22; student. June 16, 1923, first visit. Chief complaint, headache, dizziness, constant cold in nose and discharge; foul breath. Duration, four years.

Previous antrum operation, none.

Other nasal operations: Anterior end of left middle turbinate removed.

Constant cold; nasal discharge; headache, dizziness, foul breath. Vertigo, impaired hearing. Chronic hyperplastic ethmoiditis; anterior end of left middle turbinate removed. Polypoid changes in left middle turbinate.

Transillumination: Cloudy right maxillary.

Exploratory puncture: Pus, thin, granular, offensive odor.

X-ray studies: Cloudy right maxillary.

Findings in puncture following X-ray: Pus.

X-ray study following washing of antrum: Shadow on left where washed.

X-ray with lipiodol: Space along malar right side shows thickening. The lipiodol did not fill the antrum completely due to presence of pathologic tissue.

X-ray study of retention of lipiodol: None.

Operation: Right, Caldwell-Luc.

Operative findings: Mucous membrane very thick and large amount of polypoid tissue and polyp removed from the malar side of the antrum. Pus found on right side.

Bacteriology: Streptococcus; nonhemolytic.

Pathology microscopic: Fibromyxomatous tissue and polyp.

Deductions: None.

General remarks: None.

The atrophic maxillary antrum is the most perplexing of all antrums and is the one that is least suspected as the causation of disease. Although it is of the nonsuppurative type, still there are transient periods during which time serous gelatinous fluid can be found (as was pointed out by Emerson). During the period when this transient fluid is present the antrum presents certain typical physical signs, amongst which are the varying degrees of opacity on transillumination, fluid on puncture and shadow on X-ray. During this time the patient often presents a group of symptoms simulating vasomotor rhinitis, hay fever when in season, or the perennial type of hay fever and some form of allergy. On the other hand, there are many times when the serous gelatinous fluid is absent, and it is then that we are apt to overlook the antrum as a source of disturbance. This is especially true in the overdeveloped maxillary antrum with atrophy. On analysis of this type of case we note overdeveloped antrum, atrophy and thinning of the mucous membrane and absence of the serous gelatinous fluid. Upon further study, transillumination reveals exaggerated light permeability through the canine fossa under the orbit and through the conjunctiva. The antrum puncture and X-ray findings are negative. How then are we to diagnose this type of maxillary antrum? Lipiodol, when instilled into this type

of antrum, will reveal the true state of the antrum and the lack of density of the mucous membrane. However, sometimes it is difficult to judge mucous membrane and to differentiate the atrophic from the normal. Repeated radiographs taken of the antrum filled with oil will aid us greatly in distinguishing the atrophic from the normal mucous membrane, as we find that the oil in the normal antrum will be eliminated within 24 to 48 hours, while in the atrophic type the oil remains in the antrum from 10 to 12 days.

Cysts are a distinct entity in themselves, and I am only mentioning them in passing as a factor in maxillary pathology. We generally divide them according to their origin, which is either dental or maxillary. In the cysts of dental origin we have the root or radicular cyst which originates in the periodontal membrane. The follicular or dentigerous cyst starts from the enamel of an unerupted tooth.

The intramaxillary cysts are the mucous cysts, polyps undergoing cystic degeneration and hydrops of antrum of Highmore secondary to inflammation (as quoted by Hajek).

Cysts not infected may not present any nasal symptoms at all, although in certain types, especially those that are very extensive, there may be external facial displacement, palatal displacement presenting local swellings, and on palpation crackling sounds may be elicited. The smaller cysts rarely present any symptoms. Occasionally the patient may give us a history of discomfort on the side of the face that is involved. Another symptom often overlooked is the nasal polyp that may be present on the side of the antrum involved.

In our experience we found that 80 per cent of nasal polypi are associated with antrum disturbances. In the greater majority of cases, however, maxillary cysts are symptomless. On transillumination the findings are practically negative or the opacity is so slight that it is easily overlooked. X-ray may reveal a slight halo, but this also is very often overlooked unless actual bone destruction has taken place. The cystic fluid is invariably amber in color. When the fluid coagulates we know that it is inflammatory in character. On the other hand, when the fluid does not coagulate it is simply a transudate.

In this type of case, before removing the needle, we make it a rule to instill lipiodol directly into the cyst through the needle in situ, inflating the cyst with lipiodol. X-ray studies taken of the cyst when it is filled with the oil will reveal more clearly the size, position and the contrast of the surrounding structures. It is advisable to reinsert the needle in another position and force some oil into the antrum. By doing this, the X-ray will reveal any other pathology that may be present in the antrum, as the oil will cover the surface of the structures and thus cast a shadow on the surrounding area. Shall we say that sometimes transillumination, X-ray and puncture may be negative, due to the fact that the cystic fluid is so clear and the cyst may not be in the pathway of the needle and therefore may be missed? Therefore it is most essential that we obtain a history from our patient. Should the patient complain that at various intervals he has noticed an escape of yellowish fluid through the nose, then we should by all means have lipiodol instilled into the antrum, as in this type of case the oil will surround the cyst or cyst sac. Otherwise it may escape our notice when the ordinary means of making a diagnosis are employed.

For illustration, we quote a cyst case:

Case 19, X-ray 1663.

T. L., male, 28; laborer. Upper right second bicuspid extracted; two months previous patient had pain on right side of face, tenderness and feeling of fullness. Duration, eighteen months before extraction of tooth.

Pain over right side of maxillary antrum. Discharge from mouth through tooth socket.

Focal infection: None.

Clinical examination: Small sinus from second bicuspid socket with offensive discharge.

Transillumination: Cloudy right maxillary.

Exploratory puncture negative.

X-ray studies: Shadow covering most of the right maxillary antrum. Only small triangle of antrum remaining. Cyst forcing the antrum up.

Findings in puncture following X-ray: Negative.

X-ray study following washing of antrum: Shadow still remaining.

X-ray with lipiodol: Only small amount of lipiodol forced through the socket, the rest being filled up with the pathologic tissue.

X-ray study of retention of lipiodol: No study.

Operation: Single; right Caldwell-Luc.

Operative findings: Cyst; small communication into the antrum. Necrosis of the maxillary process which was removed. Communication between cyst cavity and antrum was established. Caldwell-Luc operation performed and upper right first bicuspid extracted during operation; upper right first molar extracted, the second bicuspid having been removed before.

Bacteriology: Smears; mostly blood cells, no organisms. Cultures, heavy mixed growth of nonhemolytic streptococcus and pneumococcus.

Pathology microscopic: Chronic inflammatory tissue, composed of fibrous tissue with extensive cellular infiltration but no evidences of tumor formation. No signs of malignancy.

Deductions: None.

General remarks: Dentigerous cyst.

Retention deductions: None.

SUMMARY.

In analyzing fifty operative cases in which pathologic studies were made we found hypertrophied tissue, atrophy, polyps, granulation tissue and cysts. It is a curious observation that the streptococcus hemolyticus and streptococcus viridans predominated, and these organisms were more often found in the nonsuppurative maxillary sinusitis.

It is my practice to observe conservatism in the treatment of maxillary infection, especially when it is of the nonsuppurative type. In some of the asthmatic cases, although we found nasal polypi, we at no time were able to find pus on puncture of the antra. We found that about 80 per cent of nasal polypi were associated with maxillary infection and improved after operation.

In a group of cardiorenal cases antrum punctures were negative at various times. In some cases it was difficult to force air through the antrum, due to the edema of the antral mucous membrane, polyps, or polypoid tissue around the ostium or around the needle point orifice (the needle being patulous by test). Lipiodol in these cases definitely outlines the intramaxillary pathology.

In a group of diabetics with hyperglycemia we found that the blood sugar could not be reduced until the maxillary antra were operated upon, thus removing the source of infection (Caldwell-Luc performed).

The problem of conservatism and operative interference should naturally be weighed carefully. However, from our microscopic study of specimens and by the methods enumerated we feel that no conservative maxillary operation of the intranasal type will cure a case with tissue changes after it has passed the stage of resolution. In view of the fact that we found hypertrophic, hyperplastic, atrophic, granulation tissue, polyps and cysts in the study of these cases by the methods described previously, I feel that the open canine fossa operation with complete inspection of the intramaxillary structures will afford us the best opportunity of eradicating diseased tissue.

Ventilation in early maxillary infection is important and helpful and should be instituted as soon as possible. Ventilation instituted after gross pathologic changes have taken place is of less value, as in a group of 38 cases upon whom conservative maxillary operations, including intranasal and external canine fossa operations, were performed, the focus was not completely removed, nor did the patients improve until thorough radical operations (Caldwell-Luc) were performed. I therefore, without hesitation, recommend that so long as we are operating we should remove all possible tissue in the path of operation. I would rather err on the side of complete removal than predispose my patient to secondary operation and fail in the purpose of removing the source of focal infection.

May I add that I wish to agree with Ross H. Skillern, who first pointed out to me that the antral nasal flap, which for many years was preserved and thrown into the floor of the antrum in order to stimulate epithelization, should be removed. Therefore, when we undertake to perform a radical operation in order to eradicate focal infection we now remove all mucous membrane and also include the nasal mucous membrane flap.

I wish to reiterate that nonsuppurative maxillary sinusitis has, from a focal phase, been sadly overlooked. I therefore wish to lay special stress on the fact that lipiodol here is a very great factor in our aid to diagnosis. We feel that it has helped us greatly in correlating our present conclusions.

I hope that the material here presented will serve to bring about a clearer understanding of the diagnosis of antral pathology.

I wish to take this means of thanking Drs. George M. Coates, Ross H. Skillern, John A. Kolmer and William Ersner for their invaluable assistance and cooperation.

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Fig. 1. Case 5—Before irrigation.

Fig. 2. Case 5—After irrigation.

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Fig. 3. Case 5—With oil.

Fig. 4. Case 17.

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Fig. 5. Case 17.

Fig. 6. Case 17—Before irrigation.

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Fig. 7. Case 17—After irrigation.

Fig. 8. Case 19.

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Fig. 9. Case 19.

VI.

X-RAY EXAMINATION OF DEFORMITIES OF THE NASOPHARYNX.

By Prof. Arthur Schüller, M. D.,

Vienna, Austria.

The shape and size of the nasopharyngeal cavity is determined to a great extent through the configuration of the surrounding bony structures. The anterior border of the nasopharynx is formed by the posterior edges of the two plates of the pterygoid process and of the vomer, the roof by the lower surface of the sphenoid, the posterior wall by the condyloid part of the occipital bone and the anterior arch of the atlas.

It is possible through X-ray pictures to visualize the contours of these bony structures and to form an opinion as to the dimensions of the epipharynx. The lateral view seems to be the best for this purpose, but the axial or the anteroposterior pictures also give occasionally a good view of the shape of the nasopharyngeal cavity.

The X-ray plates, Figs. 1 to 3, show transverse pictures of nasopharyngeal cavities with normal configuration at different ages. The profile of the bony surroundings of the nasopharyngeal cavity shows an arch-shaped and dorsalward convex contour. Anteriorly a shadow line can be seen that corresponds to the free edge of the pterygoid processes. (Pr). Above, one can see the projection of the base of the corpus sphenoidale and of the basilar part of the occipital bone (D), posteriorly the outline of the condyloid process of the occipital bone (Co) and of the anterior arch of the atlas (V). For the lower limit of the epipharynx we take the line drawn from the posterior edge of the hard palate (G) to the projection formed by the anterior tuberculum of the atlas. The length of this line can be taken as a measure of the transversal dimension of the epipharynx. A vertical line drawn from the highest point of the roof of the pharynx to this horizontal line gives a measure of the vertical dimension of the nasopharyngeal cavity.

Fig. 1. Lateral view of nasopharynx of girl, 14 years of age.

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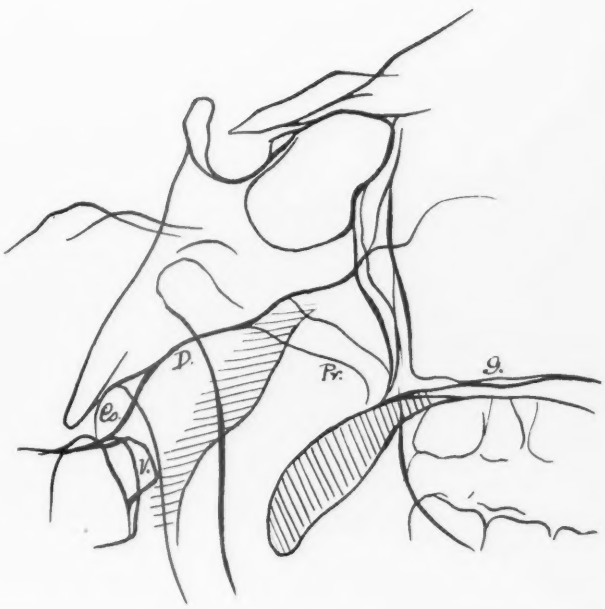


Sketch to Fig. 1. (Shadows of soft parts are shown through shading.)
Co, Condylus occipitalis; D, Roof of nasopharynx; G, Hard palate;
Pr, Processus pterygoideus; V, Atlas.

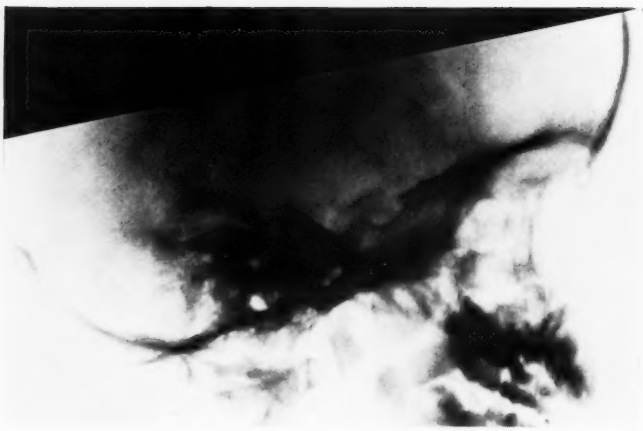
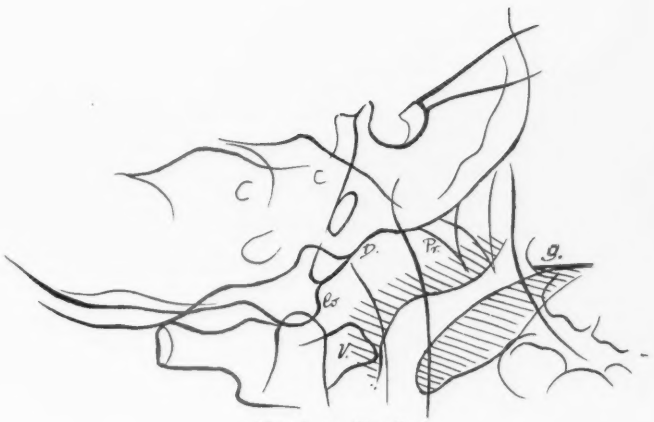
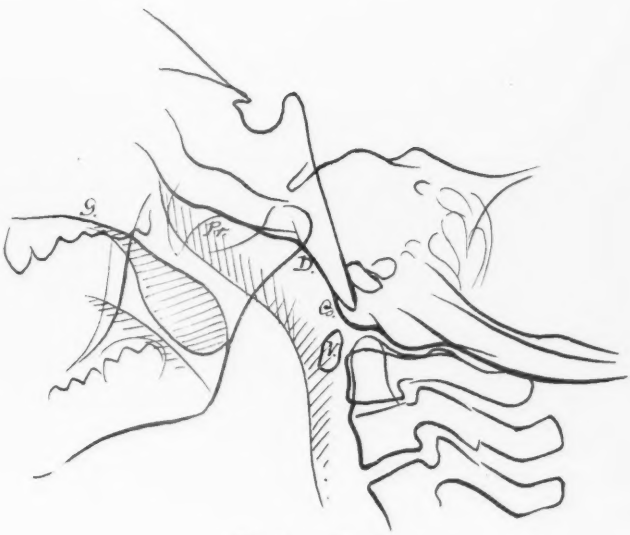


Fig. 2. Lateral view of child, 5 years of age.



Sketch to Fig. 2.

Fig. 3. Lateral view of child, 16 months of age.



Sketch to Fig. 3.

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In the area of the epipharynx one can see the projection of the rather dense shadow of the mandible, also the outline of the zygomatic process and of the tuberculum articulare of the temporal bone, finally the delicate shadows of the soft parts of the pharynx. The width of the shadow line of the soft tissues covering the posterior wall of the pharynx is about 1 cm. It gets somewhat narrower upwards and frontalwards. In case of adenoid hypertrophy there is a marked tumor-like projection into the lighter area of the pharyngeal cavity. The anterior lower border of this cavity is formed by the outline of the shadow of the soft palate which appears rather dense, due to a summation of shadows of the mandible.

The axial picture of the base of the skull is taken in the following way: The head hangs down over the edge of the table, the film is over the parietal region. The central ray traverses the intersection of the median-sagittal plane with the ear-vertical plane. This film shows details of configuration of the bony epipharyngeal walls; anteriorly the shadow of the inner plate of the pterygoid process, posteriorly the outline of the anterior arch of the atlas, and also the soft shadows of the soft parts forming the lateral walls of the epipharynx. All these details usually can be seen distinctly against the lighter area of the pharyngeal cavity.

In the anteroposterior picture of the base of the skull the film is at the occiput, the central ray running along the intersection of the medial-sagittal plane with the horizontal plane laid across the superior edge of the orbits and the ears. This view shows the projection of the base of the sphenoid bone, the contours of the condyloid processes of the occipital bone and the outline of the atlas.

Even under normal conditions the configuration of the base of the skull and the relation of the first cervical vertebra to the base of the skull may show a number of variations. Lateral roentgenograms are an important aid in studying these variations. (See Fig. No. 4.)

The length of the base of the skull is estimated from the distance of the root of the nose ("Nasion") to the end point of the clivus ("Basion") or from the distance of the Nasion to the external occipital protuberance.

The niveau-relations of the fossæ anterior, media and posterior of the base of the skull can be studied on a lateral picture by measuring the vertical distance of their lowest points from the eye-ear-plane which is taken as the standard horizontal plane, similarly as this is done on a medial-sagittal section through the skull. The plane traversing the lower edge of the orbita and the highest point of the external auditory canal (eye-ear plane) runs normally through the deepest point of the middle cranial fossa. In a plane parallel to the eye-ear plane, across the deepest point of the anterior and posterior fossæ, the vertical distances of these planes from each other will be about equal and will measure about 2 cm. in adults.

The three cranial fossæ form a sort of stairs of equal heights. The deepest points of these fossæ—i. e., the lowest point of the lamina cribrosa, the lowest point of the corpus sphenoidale and the most posterior point of the foramen occipitale are in normal skulls as a rule on a straight line. (Basal line ST.)

The curving ("kyphosis") of the base of the skull is determined by the size of the basal angle—i. e., of the angle open ventrally and downwards and formed by the sphenoid plane and the clivus on the medial-sagittal section of the skull. This angle usually measures 110°. If the basal angle approaches a right angle ("hyperkyphosis") then the nasopharynx is usually narrower in its anteroposterior diameter. In case of a large angle ("platybasia") the posterior pharyngeal wall is, as a rule, at a longer distance from the choanæ.

The relations of position of the first cervical vertebra to the base of the skull are determined by the distance of the anterior arch of the atlas from the posterior choanæ and by the difference in niveau between the plane of the arch of the atlas and the plane of the hard palate, taking the measurements in a middle position of the head in the occipito-atloid joint. Hultkrantz's exact investigations put the excursions of the occipito-atloid joints at about 20°. With these rather limited excursions the distance of the anterior arch of the atlas from the choanæ in the same individual and in extreme positions—i. e., in maximal flexion and in maximal extension of the head, is not very considerably different. It measures in both positions about 2.5 cm.

Testut and Jacob describe three principal forms of the pharyngeal arch. In the majority of cases they found the angular arch, in which the roof of the pharynx slants backward and downward and forms a very blunt angle with the posterior wall ("Nasopharynx à voûte ogivale ou à recessus"). The whole cavity is considerably above the level of the palate. The second type, found in one-third of the cases, shows the rounded arch shape. Here the roof of the pharynx rises to a medium height above the level of the palate. ("Nasopharynx à voûte cintrée.") The third type, the low arch, is characteristic of childhood. The space is low, the pharyngeal arch is at the level of the inferior turbinate, the tubal openings are at the level of the palate ("voûte surbaissée"). The anterior arch of the atlas is somewhat below the level of the hard palate.

Anomalies of shape and size of the epipharynx arise through congenital or acquired deformities of the base of the skull or of the upper cervical vertebræ. There are up to the present time only very few data in the literature about the practical importance of such abnormalities. Wetzel points out that tumors of the nasopharynx, including under this term hypertrophies of the pharyngeal tonsil, reaching a given size, will cause a more marked obstruction to nasal breathing if the nasopharyngeal cavity is narrow. McCarthy discusses the stenosis of the nasopharyngeal space due to a protruding arch of the atlas. Bernfeld describes the difficulties of complete adenoidectomy in a narrow nasopharynx and emphasizes the necessity of repeated operations in such cases. Goldhamer and Schüller review the anomalies in form and size of the nasopharynx caused by variations of the posterior cranial fossa.

Deformities of the bony surroundings of the nasopharynx may be classified in three groups. The first group includes all forms of narrowing of the nasopharyngeal cavity due to a shortening of the base of the skull. This shortening may be congenital or acquired in early childhood. An example of the congenital type is the shortened cranial base found in chondrodystrophy and is due to a premature ossification of the os tribasilare. The basal angle is decreased in this type ("hyperkyphosis").

As an example of the acquired type the so-called occipital sinking of the skull may be mentioned. This is found in rachitic, or

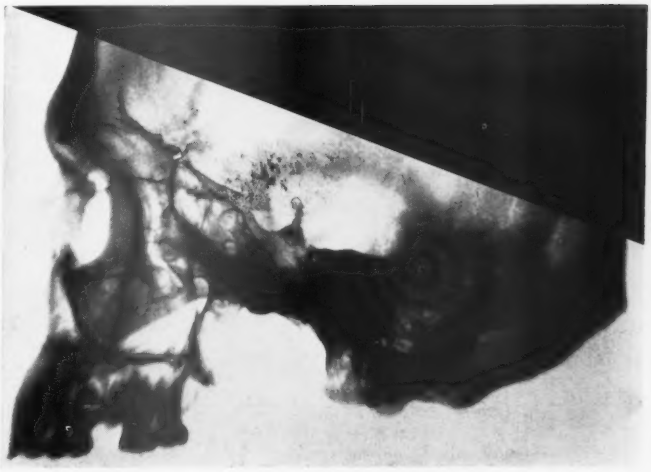
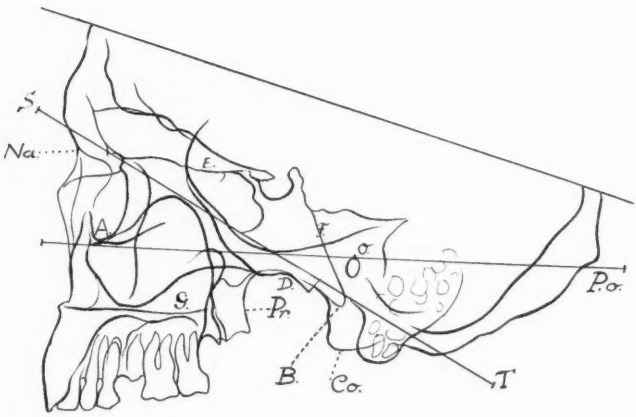
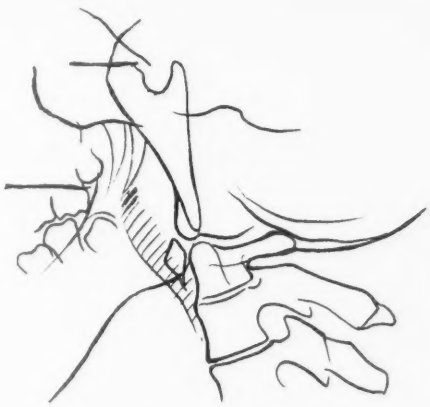


Fig. 4. Profile picture of skull halved in midline.



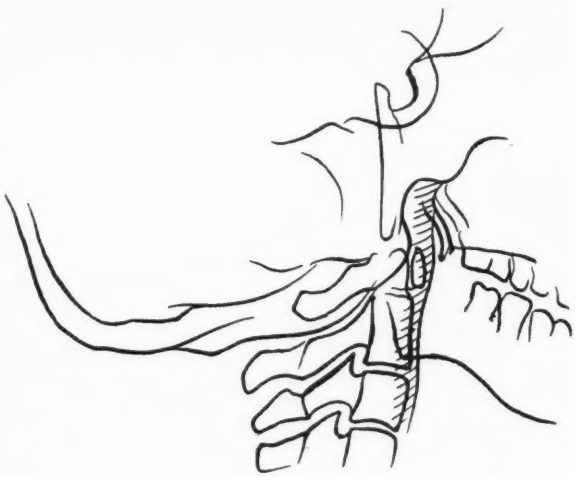
Sketch to Fig. 4. A, Lower orbital point; B, Basion; Co., Condylus occipitalis; D, Roof of epipharynx; E, Planum sphenoidale.

Fig. 5. Profile picture of pharynx of boy of 8 years, with congenital narrowing of the nasopharynx due to hyperkyphosis of the base of the skull.



Sketch to Fig. 5.

Fig. 6. Profile picture of the nasopharynx of patient—same as of Fig. 5 —at age of 22 years.



Sketch to Fig. 6.

paralyzed or mentally defective children and is due to too long continued lying on the back. In these cases the curvature of the posterior cranial fossa is accentuated, the posterior pharyngeal wall approaches a vertical direction and is closer to the anterior wall than normally.

In the second group we include congenital or acquired deformities in the region of the upper cervical vertebræ. These are the so-called atlas-assimilation and the basilar impression and the congenital luxation of the atlas on one side, the rachitic deformities (lordosis) of the cervical vertebral column and the traumatic dislocations in the craniovertebral joints on the other side. These abnormalities may cause a considerable narrowing of the nasopharynx in the sagittal diameter.

The third group consists of deformities of the nasopharynx found in craniostenosis or narrowing of the cranium due to premature synostosis of the sutures. They are caused by a change in the shape of the cranial base typical for craniostenosis—i. e., by the marked downward protrusion of the body of the pterygoid bone. This produces a shortening of the nasopharynx along its vertical diameter. A few clinical cases belonging to these three groups will be described here briefly.

Case No. 1. Congenital Hyperkyphosis of the Cranial Base.—Eight-year-old boy, under rhinologic treatment since early infancy because of difficult nasal respiration. As the cause of the obstruction a hypertrophy of the adenoids was first thought of. As repeated adenoidectomies failed to give any relief, roentgenologic examination of the nasopharynx was made. The film (Fig. 5) shows an abnormality of the shape of the cranial base—i. e., a decrease of the basal angle to nearly 90°. The deformity of the nasopharyngeal cavity as a result of this was of such a degree that there was absolutely no air space between the nasal cavity and the nasopharynx. The plane of the posterior choanæ stood at a sharp angle to the pars basilaris of the occiput; the distance between the anterior and posterior wall of the bony nasopharynx measured 1 cm.

This patient presented many other congenital abnormalities in the region of the head as caput quadratum, ankyloblepharon, progenia and atresia of the auditory canals. Therefore the

deformities of the base of the skull were interpreted as congenital abnormalities. A recent clinical and roentgenologic examination at the age of 22 years did not show any marked difference from previous findings. (Fig. 6.)

Summary of case: Complete obstruction of nasal respiration, not relieved by repeated adenoidectomy; X-ray reveals a congenital deformity of the cranial base—i. e., a hyperkyphosis.

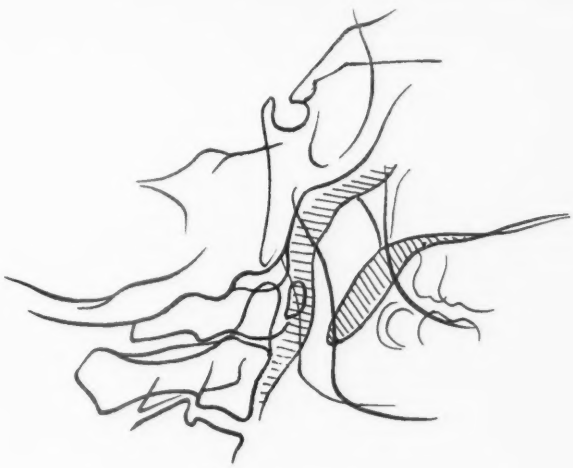
Case No. 2. Stenosis of the Nasopharynx Due to Kyphosis of the Cranial Base.—Boy, 14 years of age, an idiot. Breathing with mouth open. As the cause of the difficulty in nasal breathing an hypertrophy of the adenoids or possibly a malformation (pharyngeal meningocele) was suspected. X-ray plates were taken to find out the cause of the respiratory obstruction. They show (Fig. 7) that the bony walls of the pharynx form a sharp angle due to a kyphosis of the skull base.

The shadows of the soft parts in the area of the nasopharynx show no abnormalities. Nothing can be found to substantiate the presence of a meningocele or of enlarged adenoids. An adenoidectomy performed on request of the physician in charge of patient did not produce any adenoids of any appreciable size; only at the opening of the tubes was some adenoid tissue to be found. The nasopharynx was found to be so narrow that the roof the pharynx could be reached with only the smallest curette.

Summary of case: Mouth breathing due to narrowness of the nasopharynx caused by a kyphosis of the skull base in an idiot.

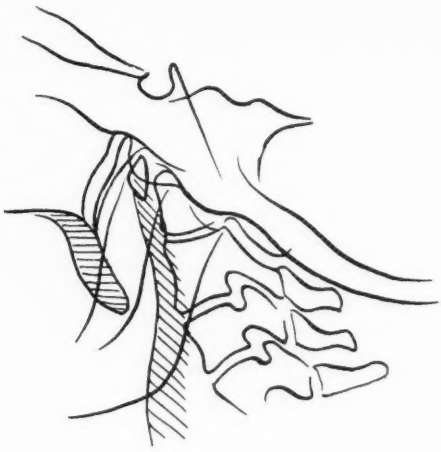
Case No. 3. Congenital Defect of the First Cervical Vertebra.—Woman, 41 years of age, whose head has been bent backward for many years, until it is lodged between the shoulders and the occiput, touches the back of the neck. Recently she began to complain of occipital headaches, and a difficulty of speaking and swallowing developed. Examination showed, besides the abnormal position of the head and a considerable shortening of the neck, a right-sided paralysis of the tongue. On the transverse X-ray picture (Fig. 8) one can see clearly a lordotic curvature of the cervical spinal column and a dislocation against the base of the skull. The head appears to be displaced several cm. backward so that the foramen magnum

Fig. 7. Lateral view of nasopharynx of an idiot, 14 years of age, with kyphosis of the base of the skull.



Sketch to Fig. 7.

Fig. 8. Lateral view of the nasopharynx in a woman, 41 years of age. with atlas assimilation.



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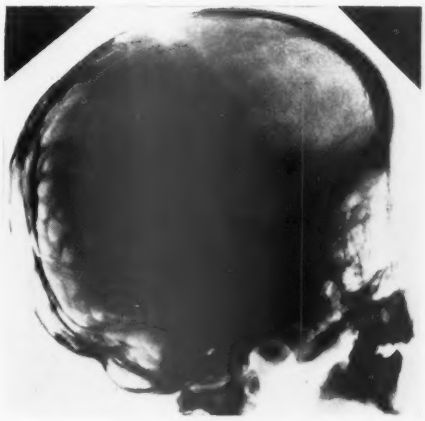
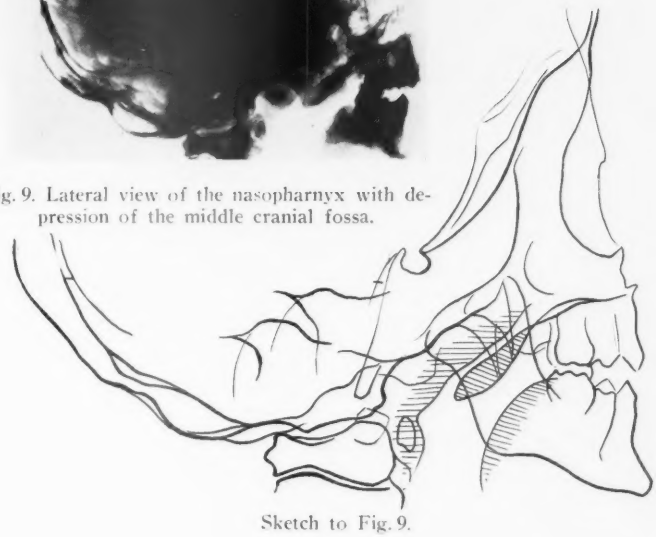


Fig. 9. Lateral view of the nasopharnyx with depression of the middle cranial fossa.



Sketch to Fig. 9.

is on top of the spinous processes of the upper cervical vertebræ. The atlas is underdeveloped. Due to the backward dislocation of the head the choanæ touch the anterior surface of the vertebræ. The nasopharyngeal cavity is almost obliterated. The history of this patient and the findings justify the diagnosis that this is a congenital malformation of the atlas and of the occiput—i. e., a so-called "atlas assimilation." In such cases there is an incomplete development of the atlas and of the parts of the occipital bone that form the edges of the foramen magnum. Occasionally also the epistropheus or even the other cervical vertebræ are incompletely developed. These parts of the skeleton are not well able to carry the weight of the head, and so it occurs frequently that patients with such congenital malformation in the course of years develop a luxation of the head. It is easy to see that such a malformation of the upper cervical vertebræ must have a decided influence upon the shape and function of the nasopharynx. If the malformation is asymmetrical then the usual clinical picture to be found is a caput obstipum congenitum.

Case 4. Narrowing of the Epipharynx Due to Low Position of the Middle Cranial Fossa.—A child, 2¼ years old, is brought to the pediatric clinic with the history that it has severe dyspnea, particularly at night. The breathing of the patient is labored and noisy, even in the waking state. Transverse X-ray picture of the head (Fig. 9) shows a hypocephalic skull with very marked impressiones digitatæ in the region of the occipital bone. This is probably due to a pressure of the weight of the brain, principally on these parts, caused by long continued lying on the back. The base of the skull is considerably deformed. The floor of the middle fossa is markedly low in relation to the level of the lamina cribrosa, but it is almost on the same level with the floor of the posterior fossa. Due to this depression of the middle fossa the roof of the epipharynx approaches the soft palate so nearly that the soft parts almost completely touch each other and obstruct the passage of the air, particularly during sleep, when the strongly developed uvula falls backwards.

Summary of case: Very marked obstruction of nasal respiration due to low position of the pharyngeal roof caused by congenital deformity of the skull.

CONCLUSIONS.

X-ray examinations in cases of difficult nasal respiration occasionally reveal as the cause of the trouble deformities of the base of the skull or of the upper cervical vertebræ, particularly hyperkyphosis of the base of the skull, atlas assimilation and depression of the middle fossa in craniostenosis. Orthopedic treatment may in some instances correct the deformities and relieve the nasal obstruction.

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VII.

THE DEVELOPMENT OF THE SINUSES AFTER BIRTH*

By T. E. Carmody, M. D.,

Denver.

While there seems to be abundant literature treating of the subjects of focal infection from the regions of the upper respiratory and digestive tracts, and while this literature has added much to our knowledge of disease as it pertains to our familiar line of endeavor, we have apparently not touched the depths.

We do not wish to belittle the excellent work which has been done by the pioneers and their followers, but it seems that there is much to be accomplished in prophylaxis.

It is almost impossible to find an adult in civilized life who has no infection of sinuses, teeth or tonsils, even though many have been deprived of the latter in childhood.

We say without fear of contradiction that the infant who has reached the age of one year without the so-called acute cold does not exist. Apparently we do not realize what an acute cold is? In our opinion every acute cold involves not only the nasal mucosa but that lining the sinuses as well.

Taking this as a premise, which, however, may be questioned, how long or how many separate attacks must any individual have before evidence of chronic change, either in the membranous lining of a sinus or in the bone itself, takes place. We believe this may depend on individual variations which may be due to susceptibility, virulence of infection, etc. In some cases only one attack occurs, in others, many.

Roentgenologic evidence of pathology in the sinuses may not be present in the early stages of infection, and this was a

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From the Child Research Council, University of Colorado, School of Medicine, Denver, Colorado.

source of error and much controversy until very recently, and there are many debatable points as to extent and change in chronic cases at this date.

From the standpoint of the radiologist, the X-ray portrays pathology according to the interference with the passage of these rays through the tissue. The question naturally presents itself as to how much pathology must be present to obstruct the rays, or, in the case of rarefaction, to offer lessened resistance to their passage.

If we consider the lining membrane of the nasal cavity or of the accessory sinuses in the rôle of culture media, we may find a few colonies scattered over the membrane or possibly only one colony gradually spreading until sufficient surface is involved to produce visible exudate. If resolution takes place, for any reason, at this time, the tissues may radiograph as normal, but should the infection progress into the epithelium, subepithelial tissue, or even to the periosteal covering of the wall, a different picture will result, even though there may be no involvement of the bone.

The clinical and radiographic findings should agree, and usually will if careful study is made. Density of different portions of a sinus may be accounted for by destruction of portions of the lining membrane, while others may remain normal or but little changed. If the theory of Wittmaack[1] accounts for change in the mastoid or interference with development, the same may surely be true of bone cells in other parts. However, in order to determine either we must prove that the osteogenetic layers of the periosteum are affected. Function is merely interrupted in early inflammatory reaction but is entirely changed in the subacute or chronic, due to destruction of functioning tissue.

Many of us recall the controversy regarding dental and nasal infection in maxillary sinus disease. With the evidence accumulated in our work in the Child Research Council, which is a continuation of the work begun under the Selmene Winter Foundation, together with that shown in operative cases by Shea, such a discussion can, we believe, be almost entirely eliminated in patients under twenty years of age. Furthermore, the suggestion which has been followed in cases of enteritis

in children, apparently due to inflammatory process in the middle ear and mastoid, ignoring entirely the original source of infection, is founded upon the wrong premise. Not that absorption may not take place from middle ear and mastoid, but we believe and have proven to our own satisfaction that the primary infection is, in the overwhelming majority of cases, in the nasal passages and sinuses.

It is barely possible that a small number of cases may be of hematogenous or lymphogenous origin, but as in the case of any middle ear infection in the adult, this is acknowledged as rare. We wish first to try and prevent any infection, and to do this we must begin with the infant.

Much effort has been made and with excellent results in teaching the young mother to sterilize food and utensils, looking to a better intestinal condition. On the other hand, little has been done by the pediatrist or rhinologist to prevent the mother, father or nurse from infecting the respiratory apparatus.

We have found many children with respiratory infection whose mothers, fathers and nurses were apparently healthy, but we have not found a child whose mother had sinus disease or pulmonary infection who was free of both upper and lower respiratory infection. There may be such cases, for there may be mothers who will not deliberately pass the infection to the child's mouth or nose.

Infection in the sinuses is much more pernicious than any other focal infection, with the possible exception of the tonsils, and in both for the same reason, because absorption of toxin may be supplemented by ingestion as well. Drainage from tympanic cavity through eustachian tube is not so important a factor, although this must be kept in mind.

It seems quite possible, as suggested by us, that infection of sinuses may affect the so-called ductless glands, as has been accepted in the case of the thyroid as related to tonsil infection. (Shurley.[2])

Only recently three cases of enlarged thymus were traced to infected maxillary sinuses in infants, and in all recession took place after draining these sinuses. These were found in

private cases. The author suggested this possibility in the discussion of a paper by Dr. Wasson on "Thymic Stridor," read before section of pediatrics, American Medical Association, in 1927, and subsequent results have proven this correct.

STUDY BY PHYSICAL EXAMINATION, VARIOUS LABORATORY TESTS AND RESULTS NOTED.

After this, radiographs of sinuses and chest are made in all cases, and if thought necessary other parts. These findings are then taken and correlated and considered in connection with environment, food, pathologic history or lack of same in the parents. It has been manifestly impossible, as noted above, to have all of these infants from normal families or from the same social strata. Neither has this been desirable, for obvious reasons.

While these results have yielded much information, special cases, as seen in private patients and those seen in outpatient clinic at Children's Hospital, have been used to a certain extent as controls.

From the standpoint of the sinuses, and this probably applies equally to other parts as well, we have been able to follow the development of these cavities in health and disease. Autopsies have tended to corroborate our findings, but when not, special notation has been made. Operations can be used as the same control as autopsy only when radical enough to expose large areas of mucosa for histologic examination which is not advisable in very young children and infrequently in older ones.

The course of development of the sinuses is definite in normal infants and young children, but, as above stated, many things must be considered besides infection, many of which are given more weight than they deserve, and many less, depending on the bent of the investigator. Furthermore, it is difficult to decide cause and effect.

The glandular factor in development must be active from the moment of birth, while the food factor is delayed at least hours, and what effect may these have on infection of the respiratory tract, which may of necessity begin with the first inspiration because of the possibility that the first air inspired contains bacteria which may emanate from the mother, physician or nurse?

We cannot take up along this line the work of Dean,[3] Daniels[4] and many others which would be of interest. As before mentioned, it is not possible to determine how soon after the introduction of infectious material radiologic evidence of its presence appears, but we have learned that when we find clinically that there is a suspicion of nasal infection and a history of discharge, for even a day or two, this is corroborated by radiograph in about 90 per cent of the cases, which is higher, we think, than in adults.

The fact remains that infection of maxillary sinuses and ethmoid cells is present very early, having been found by us as early as the ninth day.

Furthermore, we believe that interference with development of the frontal sinuses is due to infection, whether this may be dependent on glandular action, food, environment, etc., or whether changes in the above may have been responsible for the first variations from normal.

CONCLUSIONS.

1. The infant does not exist who has reached the age of one year without the so-called acute cold.
2. The number of attacks and duration of trouble before a chronic sinus change takes place depends upon individual variations, due to susceptibility, virulence of infection, etc. In some cases only one attack occurs, in others many, before sufficient change takes place to be noted radiographically.
3. Infection early in life of maxillary and ethmoid sinuses interferes with development of frontals.
4. Infection of surface of mucous membrane will not cause a radiographic shadow, while deeper infections will.

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4. Daniels, Amy: J. A. M. A., September 8, 1928, p. 828.

Fig. 1. Baby B. Normal birth at term. Cleft lip and palate.
a—3 weeks, sinuses infected.

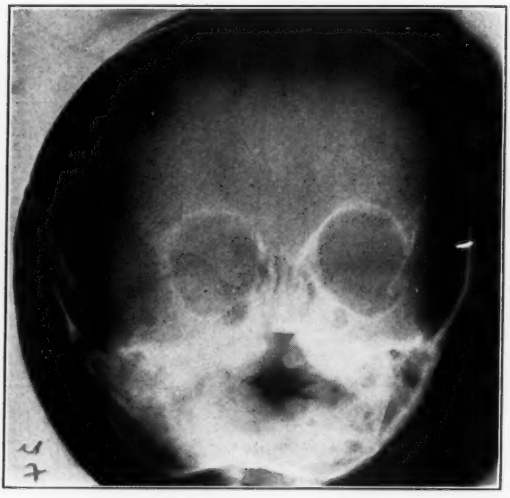

Fig 1. b—9 weeks, slight clearing.

Fig 1. c—13 weeks, slight improvement over 9 weeks.

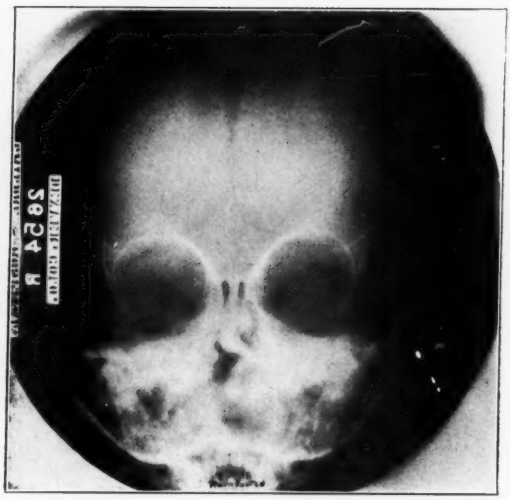

Fig. 1. d—9 months, reinfected.

Fig. 1. e—2 years 6 months, clear.

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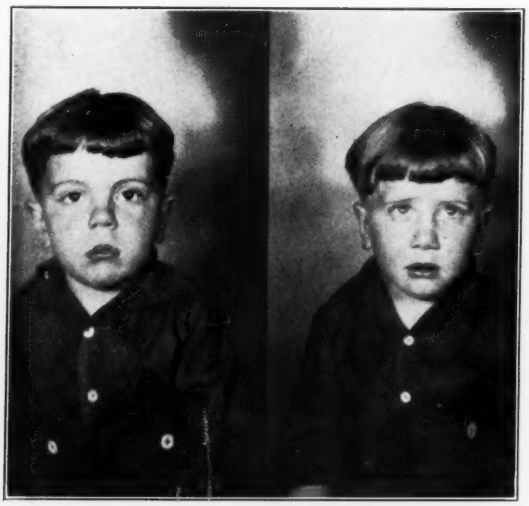

Fig. 2. S. Twins—Showing differences in development.
a—Left, J. Right, B.

J. S. (last).
June 28, 1928: 3 yrs., 3 mos., 29¼ lbs.
Birth weight, 7 lbs., 4 ozs.
Low forceps delivery; breastfed.
History of many colds and measles.
Left septum deflected.
Sinus involvement upon every exam.
Tonsillectomy recommended.
Taller than twin brother.

B. S. (born first).
June 28, 1928: 3 yrs., 3 mos., 29¼ lbs.
Birth weight, 5 lbs., 6 ozs.
Normal delivery; formula fed.
History of many colds and measles.
Sinus involvement upon every exam.
Tonsillectomy recommended.
Decaying teeth.
Soft systolic mitral murmur at 9 mos., which has apparently disappeared upon later exam.
Short, rather broad child.

Fig. 2. b—Profile of same.

Fig. 2. c—Sinuses of J. at 3 years 3 months. Involvement upon every examination. Septum deflected left.

Fig. 2. d—Sinuses of B. at 3 years 3 months. Involvement upon every examination.

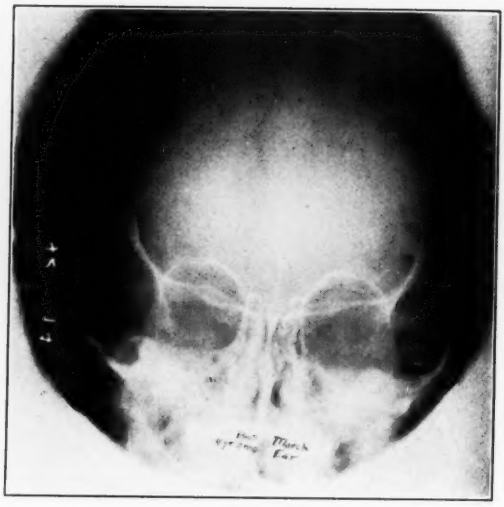

Fig. 3. a—R. L. Sinuses at 4 years 3 months. Infection.

Fig. 3. b—J. R. Sinuses at 5 years 6 months. Clear.

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Fig. 3. c—E. Y. Sinuses at 6 years 6 months. Slight cloudiness of ethmoids and maxillary sinuses.

Fig. 4. a--R. F. 8 years of age. Acute sinuses. Frontals present.

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Fig. 4. b--J. S. 9 years of age. Chronic sinuses. Frontals absent.

Fig. 5. a—A. K. Sinuses at 12 years. Frontal sinuses undeveloped as a result of chronic infection of maxillaries and ethmoids.

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Fig. 5. b—L. K. Sinuses at 12 years. Frontal sinuses developed. First acute infection at 12 years.

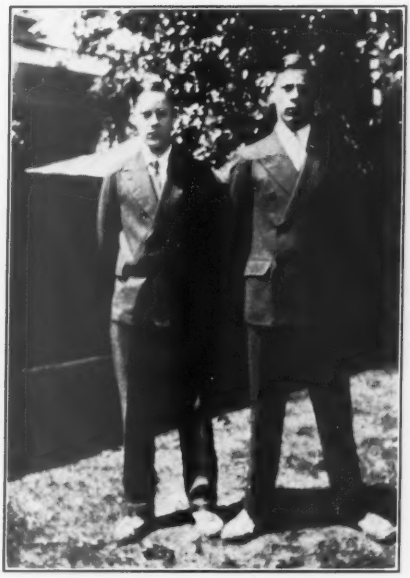

Fig. 5. c—Photo of K. boys. Underdevelopment of A., which may be due to the chronic sinus infection or endocrine imbalance.

VIII.

SOME PRACTICAL CONSIDERATIONS IN THE PROBLEM OF SINUS INFECTION AND DRAINAGE.*

By Harry L. Baum, M. D.,

Denver.

It is frequently stated that man's evolution into the erect position has so placed the ostia of his sinuses that they are predisposed to disease. Such thories seem to have gained credence quite generally among rhinologists, and yet I am thoroughly convinced that they lack scientific foundation.

As a matter of fact, very few of the lower animals keep the head in a position radically different from our own. Our nearest of kin, the ape, maintains his head as erect as ours, and I have observed that even while at rest many animals so place the head that their cavities bear the same relation to space that ours do when erect. Thus a dog frequently stretches his muzzle flat upon the ground, and a cat assumes a similar position, as do also many others of the lower animals. From these and other observations, too numerous and elementary to mention, I am led to believe that the lower animals have but little advantage of us in the matter of sinus drainage. The variations in the position of the head in the normal activities of life to a great extent equalize the effects of gravity in drainage of the accessory cavities. In addition, drainage of a cavity through a comparatively small aperture, not permitting the free ingress of air to compensate for the egress of fluid involves obvious difficulties, even though aided by posture and the force of gravity. Especially is this true when the contained fluid is thick and viscid, such as mucus or mucopus, and Proetz, in a recent article,[1] calls attention to facts which indicate that the presence of fluids of high viscosity practically nullify the effects of

*Read before the Mid-Western Section meeting of the American Laryngological, Rhinological and Otological Society, January 12, 1929, Denver, Colo.

gravity in sinus drainage. It is, therefore, the writer's opinion that the advantages of posture are probably in most cases negligible, when considered in the light of the physical principles involved.

On the other hand, studies in the histopathology of the nose and accessory sinuses have impressed me deeply with the importance of ciliary action in sinus drainage, and I am even more deeply impressed by the fact that most rhinologists apparently ignore this function. No thoughtful student can fail to realize that these tiny hairlike processes, which so completely cover the entire respiratory mucous membrane, must be an essential factor in its physiology. Yet the question of gravity drainage is stressed much more frequently in the literature than that of ciliary action. This must be due to the fact that mechanical means alone will suffice when the cilia are overwhelmed by large accumulations in the cavities or by other extensive disease, but nevertheless it is well to keep in mind first principles, and it is for that reason that this paper has been written.

The important factors of immunity, environment and all that they include cannot be ignored when considering a subject such as this, but their scope is too broad for the limits of these pages, and it is intended to consider here only local conditions within the nose and sinuses themselves.

Experience has shown that the frequency of involvement of the sinuses is in the following order: Antrum, anterior ethmoids, frontal, posterior ethmoids and sphenoid. These facts cannot be made to coincide with the postural theory, for although the ostia of both the antrum and the sphenoid are supposed to be poorly situated for drainage and the antrum is the most frequently diseased of all the cavities, the sphenoid, on the contrary, is the last in frequency of involvement. The frontal, though in the best position for postural drainage, is more frequently diseased than the sphenoid, while the anterior ethmoid, placed in second best position for postural drainage, is second only to the antrum in frequency of involvement. I believe, however, that this frequency of involvement can be explained on more logical and scientific grounds. The ostium of the antrum is the largest of any of the sinuses, and opens

into the lower part of the infundibulum. An accessory ostium is frequently present, larger and more posterior. These larger ostia with their favorable location make the antrum more easily accessible to any infection which may be active in the nose. An additional factor which adds to the frequency of antral infection is that of dental origin. It is admittedly responsible for at least 15 per cent of such cases, and by some authorities is quoted at much higher figures.

The anterior ethmoid group is frequently involved secondary to antral disease. The diseased antrum emptying into the middle meatus causes infection of the mucous membrane of that region. The resultant swelling interferes mechanically with drainage from the anterior ethmoid cells and predisposes to the development of disease within them. In addition, the relatively large amount of mucous membrane lining the ethmoid cells and its looser subepithelial structure makes those cavities more subject to the edematous change which is such a frequent manifestation in the nose and accessory sinuses. This, in turn, interferes with ciliary action, obstructs drainage, and predisposes to the development of infection. Also, the anterior ethmoidal ostia are advantageously placed for the rapid extension of infection from the lower chambers of the nose into the cells. Those who have carefully observed the course of suppurative disease in the antra and ethmoids have been struck by the great frequency with which ethmoid disease will disappear spontaneously following antral drainage. This has been to me strong evidence in favor of the opinion that suppurative disease of the ethmoid is very frequently secondary to antral infection, in spite of the commonly accepted theory that frontal and ethmoid infection, draining into the middle meatus, enters the maxillary ostium and thus involves the antrum secondarily. It is extremely difficult to introduce fluid into an air-filled cavity through a small opening, and with the ciliary current at the antral ostium working powerfully against it I feel that purulent secretion is probably not as frequently introduced into the antrum in this way as we have been taught to believe.

The frontal sinus has a great advantage over the other sinuses in the greater length, smaller size and comparative tortuosity of the nasofrontal duct, which operates as a protection against the entrance of infection. An additional protection is

the position of its ostium, high up in the anterior portion of the middle meatus, in the recessus frontalis. On the other hand, the frontal, once infected, is admittedly more difficult to cure, in spite of the fact that gravity is supposed to be to the advantage of its drainage. That this theory of anatomic isolation is of some value here may be deduced from the fact that all of us have observed the relatively greater frequency of frontal sinusitis during acute nasal infection in those who have had anterior turbinal resections and ethmoid exenterations, in contrast to the incidence of such involvement in noses not previously so operated upon.

For practical purposes the posterior ethmoids and the sphenoid may be considered together in this connection. Here infections are definitely less frequent than in the antrum, in spite of the fact that the sphenoidal ostium at least is no more advantageously placed for gravity drainage than the antral. But the more remote position of the ostia of these cavities makes them less accessible for easy invasion by pathogenic organisms from the nose. Thus we may explain the varying frequencies of involvement of the different cavities, regardless of the effects of posture and gravity, and thus may we conclude that these factors are not of primary importance either in facilitating the entrance of disease or encouraging its persistence within the sinuses once it has become established there.

Of much greater importance in the study of the problem of sinus infection and drainage is the question of ciliary action. Normally, cilia are present upon every square millimeter of surface of the mucous membrane of the nose and sinuses. That ciliary movement produces a current toward the ostia from within the sinuses and toward the choana from within the nose has been fully demonstrated by Yates[2] and Phillips.[3] This is a definite and purposeful action, and its object is that of clearing the mucous surfaces of secretion and dēbris. It is the primary factor of sinus cleansing and is sufficient to purge the cavity of most of its incumbrances, normal or pathologic. Thus do the cavities literally sweep their own floors, a function which cannot be satisfactorily performed by any other agency. However, the location of the cilia upon the surface and their extremely delicate structure makes them very susceptible to the effects of disease and frequently results in an

inhibition of ciliary activity or in an actual loss of cilia over large areas. Inflammatory states and the edematous change so frequently seen in the mucous membranes, with the accompanying alterations in the character of the secretions, are potent influences in slowing up and interfering with ciliary action. Pathologic conditions of long standing, especially those wherein pressure is a factor, often result in an entire loss of cilia with a more or less complete denudation of the surface. Thus we may have a changed character of secretion, an increased amount of secretion and a lessened activity of the cilia to account for accumulations in the sinuses, and in addition we may have swelling of the mucous membrane at the ostium with obstruction and increased interference with ciliary action at this important location. And here should be more specifically mentioned an influence of great importance, having, presumably, no direct relation to infection. I refer to the edematous change referred to above, so commonly met with in the membranes of the cavities contiguous to the nose. Suggesting allergy in both gross and microscopic appearance and in the condition with which it is so frequently associated, its exact cause and cure are as much a mystery today as when it was first observed and described. Regardless of its cause, its mechanical effects are easy to understand, and when conditions such as this and the others previously referred to have resulted in retention it is obvious that ciliary action alone may not be enough to completely clear the cavity. Especially is this true when the ostium is closed or when disease has destroyed or interferes with the action of the cilia. Then must we render assistance in order to remove, if possible, the physical handicap which has been imposed upon ciliary function.

Our first consideration, in this connection, should be that of restoring or preserving as nearly as possible the normal anatomic relations within the nasal cavities. This may require preliminary nasal surgery, followed by shrinkage and irrigations for the removal of accumulations in the region of the ostia. Suction, unless combined with irrigation, has always seemed to the writer to be unscientific. Fluid cannot be withdrawn from a cavity unless provision is made for the entrance of air to take its place, and in addition suction tends to increase congestion with consequent obstruction at the ostia. Inevitably,

intranasal treatment must often be supplemented by proper surgical procedures, directed to the sinuses themselves. Puncture and irrigation or irrigation via the ostium may be successful, as the first step of such treatment, by removing accumulated secretions of which the cilia are unable to rid the cavity, and which may act as a foreign body. Following this simple procedure the cilia are frequently able to perform their function unhampered without further necessity for surgical assistance. When more definite surgical intervention is required it should be as conservative as the circumstances will permit and should be calculated to provide adequate outlet for secretion and relief of pressure so that the normal function of the cilia may be restored. Actual removal of lining membrane or destruction of normal anatomic relationships within the nose should never be resorted to, if possible to avoid it, as it is remarkable how completely an apparently hopelessly diseased membrane may regenerate itself and renew its normal function, and it is, in my opinion, almost an axiom that the normal anatomic relationship within the nose is more protective against future invasion by pathogenic organisms than any abnormal condition surgically produced.

Although not intended primarily as such, this paper must appear in the light of a plea for conservatism in sinus surgery. If it does, I am willing that it should be so judged, as with such an attitude on the part of the majority of our colleagues we will hear less criticism of sinus work by the layman, a criticism which, I believe, is heard today more frequently than any other in the realm of medicine.

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IX.

NITROUS-OXID-OXYGEN ANESTHESIA FOR TONSILLECTOMIES.*

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This is a brief summary of personal experience and observations based on approximately 6,000 cases of tonsillectomies where nitrous-oxid-oxygen alone was used for the anesthetic and approximately 10,000 cases where nitrous-oxid-oxygen-ether was employed. This paper will deal with nitrous-oxid-oxygen alone except where comparisons are made with nitrous-oxid-oxygen-ether.

In any operation where a general anesthetic is given there are three individuals directly concerned, namely, the patient, the surgeon and the anesthetist. The welfare of the patient should have first consideration. Everything should be for his safety and comfort. He is entitled to the least harmful and most agreeable anesthetic if it is consistent with safety. In the selection of the anesthetic safety should come first and comfort second.

No one would jeopardize his life to avoid a day or two of nausea and vomiting or to escape an unpleasant induction with a disagreeable anesthetic. However, if these undesirable conditions can be eliminated it is greatly appreciated by the patient. He should be given a thorough preoperative examination and preparation in order to be in the best possible condition to meet the extra strain upon his physical resources due to the anesthetic, trauma, loss of blood and partial starvation.

The surgeon is in charge of the conduct of the case, assumes responsibility for the operation and usually for the anesthetic. He should have conditions obtain which will enable him to perform the operation to the best advantage. The requirements of surgeons differ in throat operations as to the degree of

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relaxation. Some demand a throat that is thoroughly and continuously relaxed. For them ether should be added to the nitrous-oxid and oxygen. Other surgeons who do equally good work require little if any relaxation, and for them nitrous-oxid-oxygen alone is all that is usually required. The continuous and complete relaxation of throat muscles is not always possible with straight nitrous-oxid-oxygen anesthesia. This may be obtained in about one-fourth of cases where no preliminary narcotic is given, while in the other three-fourths the relaxation will be intermittent or as occurs in a small percentage absent altogether. When morphin is given throat relaxation can be obtained in a much higher percentage of cases.

The surgeon who will tolerate an occasional slight interruption in order to regain the desired depth of anesthesia can work with nitrous-oxid-oxygen with entire satisfaction.

The qualifications of the anesthetist are more important than with any other anesthetic, as nitrous-oxid-oxygen is dangerous in the hands of a novice, while it is perhaps the safest in the hands of one qualified to administer it. A medical education is desirable as a foundation, as medical knowledge and experience is often a great help in safeguarding the life of the patient, especially in poor risk cases. One requirement is alertness, because changes occur quickly with nitrous-oxid-oxygen anesthesia and prompt action is necessary at times. Delay in appreciating the condition of the patient may spell disaster. Experience with other anesthetics does not qualify one to administer nitrous-oxid-oxygen, because the signs of danger are different. One who has had years of experience in the administration of a slowly acting anesthetic, such as ether, is not accustomed to rapid changes, and his experience may be more of a detriment than a help. There is no other anesthetic, the success of which is so dependent upon close attention to small details as nitrous-oxid-oxygen, and an anesthetist who is not willing to devote enough time to seeing that everything is as it should be before starting the anesthetic is not likely to make a success of its administration, especially for throat operations.

THE APPARATUS.

The apparatus should be capable of delivering a large volume of the gases to meet the requirements of mouth breathers, and

it should be so constructed that pure oxygen can be forced into the lungs should occasion require it. The writer prefers a McKesson apparatus which has been altered in three respects: First, an arrangement has been attached to the apparatus so that the maximum flow of nitrous-oxid can be instantly produced by stepping upon a foot lever and the flow of oxygen increased by pressing down a projecting bar with the hand or foot. In fact, the volume of the gases can be altered at will by the foot alone, leaving the hands practically free to assist the surgeon if necessary. Second, the small rebreathing bag has been replaced by a large one, so that the anesthetist can tell at a glance whether or not the patient is breathing through the nose.

If he is, a much smaller volume of gas is required. Third, an adaptor, with a large opening in the side, has been inserted between the ether chamber and the breathing tube so that a larger volume of gas can be carried directly into the mouth through the rubber tubing and mouth hook. This lessens the blow of which surgeons complain.

The care of the apparatus is important in order to avoid leaks. The rubber bags must be in good condition, as they are subjected to considerable pressure, and a tear in one of them during operation will seriously interfere with the anesthetic. It is essential that there is enough nitrous-oxid and oxygen on hand. Large cylinders, especially of nitrous-oxid, should be used, as the smaller ones are much more likely to freeze. A hot water bag placed on the reducing valve usually prevents freezing if the large cylinders are used. If freezing occurs hot water is poured over the regulator.

ADVANTAGES AND DISADVANTAGES.

From the patient's point of view, nitrous-oxid-oxygen anesthesia is the pleasantest, both for induction and recovery. In certain pathologic conditions, such as pulmonary lesions or nephritis, nitrous-oxid-oxygen is especially indicated. Ethylene, which is a close competitor of nitrous-oxid, is an explosive anesthetic. In tonsil operations where electric motors are used and the room filled with the vapor of ethylene, the hazard of explosion is increased. Nitrous-oxid-oxygen, although escaping into the operating room in considerable amount as used

for tonsillectomy, is entirely free from danger of explosion. The anesthesia is not likely to be so deep as to allow the inhalation of infected material with the subsequent production of lung abscess. However, no anesthetic should be pushed to the extent of abolishing the cough reflex. The writer has adhered strictly to this rule and has never had a case of lung abscess. When a patient coughs during a straight nitrous-oxid-oxygen anesthesia it is due to some blood or other foreign matter getting into larynx or trachea, at which time the anesthetic should be made lighter until the patient has succeeded in expelling it. One disadvantage of nitrous-oxid-oxygen anesthesia is the blow, which often spatters the face of the surgeon with blood. This is more likely to occur when the suction apparatus is not working properly. The anesthetist, luckily, is usually out of range.

While nitrous-oxid-oxygen anesthesia may at times temporarily inconvenience the surgeon and call for a certain amount of patience, yet even for the surgeon it has a bonus to offer, as he is delivered from the inhalation of ether vapor. If ether is used he is forced to inhale a considerable amount, which, if he has several cases in succession, may make him feel groggy and be detrimental to his well being. His office patients, whom he treats directly after his operative work, also appreciate it if no ether is used.

PECULIARITIES OF NITROUS-OXID-OXYGEN.

Nitrous-oxid-oxygen anesthesia is peculiar in this regard, namely, that it is often difficult, especially for a beginner, to tell whether the patient is too light or too deep. The same phenomena occur at both extremes. When a patient is too deep, entering the danger zone, he may move, groan, moan, utter a sharp piercing cry, open his eyes widely, vomit or the throat muscles may become spastic and the surgeon, if he is operating, will ask for deeper anesthesia. As these signs may also occur when the patient is in too light anesthesia, approaching consciousness, it is necessary to make a differential diagnosis. If the anesthetist is in doubt he should always give oxygen. In making a differential diagnosis, the color sign, while important, is not always reliable, especially under artificial light, as is used during tonsillectomy. Even with good

daylight the color is sometimes misleading. For example, a patient with pernicious anemia will not exhibit any cyanosis, even when the oxygen has been almost entirely removed from the blood. The reason for this is the small amount of hemoglobin in the circulating blood. On the other hand, when a patient has an increase of red cells above normal, cyanosis may be quite marked, even when sufficient oxygen is being given to do away with all other anoxemia symptoms. However, these examples are unusual. For the usual run of cases the writer believes in maintaining at least a fairly pink color.

MUSCULAR SIGNS.

Although the color is important, the muscular signs are more so. In the past too much emphasis has been placed upon color to the neglect of the effect of anoxemia upon the muscular system. The convulsive jumping about of a chicken after its head has been cut off illustrates the effect of the lack of oxygen.

A novice might decide that the chicken needed more nitrous-oxid instead of more oxygen. When giving nitrous-oxid-oxygen the muscles of the patient may be affected in a similar manner if the oxygen percentage has been reduced too much. Lack of oxygen may bring about the following effects: Movements of the arms, legs or face; the patient may apparently be trying to turn over or he may assume the opesthotnus position. If in a dental chair the legs, thighs and body may stiffen and it will appear that the patient is consciously endeavoring to slide out of the chair. The muscles of the chest will become progressively more spastic, making the exhalations of longer duration than inhalations, until eventually respirations will cease, usually after a very prolonged exhalation. The vocal cords contract, and as the air from the lungs passes through them phonation occurs, such as a moan, a groan or a cry.

When the muscles of the eyelids contract, due to anoxemic spasm, the eyes will be wide open. If the eye muscles alternately contract and relax, winking occurs. Sometimes the eyes occupy the central position but are usually drawn either downward, upward, inward or outward, as one set of eye muscles often becomes spastic before the others. Owing to the marked drawing of the eyeballs from the central position it may be

difficult to observe the pupils. In marked anoxemia the pupils will be widely dilated. As a rule, they are a reliable guide to the depth of the anesthesia, but it sometimes happens, though rarely, that the breathing will stop before the pupils have dilated. This is more liable to occur if morphin has been given. The heart may become irregular. Usually the rate is slower at first, but if the anoxemia is not corrected it will become rapid. The contraction of the muscles of the arteries will cause a rise in blood pressure, but later, if oxygen is not given, there will be a drop in the pressures.

In very young children, usually up to three years of age and sometimes in older children, a lack of oxygen is likely to produce a jactitation of all the muscles of the body but especially those of the face. The twitching and jerking of the muscles may continue for a minute or longer after oxygen has been freely administered and the color bright pink. With the exception of the jactitation in children, all the distressing signs of anoxemia quickly disappear after giving a breath or two of pure oxygen and the patient relaxes.

The pupils contract, the eyeballs return to a central position, the heart rate becomes normal and the respirations resume their regular rhythm. The automatic breathing may not be resumed immediately, due to flooding the blood with oxygen, and this interval, lasting from a few seconds to a minute, may make the surgeon feel concerned, although the patient is fully supplied with oxygen and his condition good.

A few years ago McKesson made the discovery that muscular relaxation could be obtained if the oxygen and other non-anesthetic gases of the blood were replaced by nitrous-oxid and then enough oxygen added to keep the patient in good condition and to do away with any signs of anoxemia upon the muscular system. He accomplishes this by what he terms primary and secondary saturation. Primary saturation consists in administering pure nitrous-oxid until the first signs of anoxemia occur. This is usually a slight twitching of the muscles about the eyes. At this time 10 or 15 per cent of oxygen is added to the nitrous-oxid, after which the twitching or other signs of lack of oxygen will disappear after a few inhalations. Secondary saturation consists in following up the primary saturation with further administration of pure nitrous-oxid until the pupils dilate or other signs of marked anoxemia

occur, such as prolonged exhalations, when a breath or two of pure oxygen is given. After the secondary saturation the administration of nitrous-oxid is continued with the percentage of oxygen added that is judged to keep the patient in good anesthesia. Sometimes it is necessary to repeat the secondary saturation two or three times in obstreperous patients. The writer has followed this method for the general run of his anesthetics but has not found secondary saturation necessary in the majority of the tonsil cases.

The range of anesthesia varies greatly with different patients. Some can be maintained in good anesthesia with an oxygen percentage varying from 10 per cent to 25 per cent. That is, they will be quiet and relaxed, whether the oxygen is reduced to 10 per cent or raised to 25 per cent. With others a variation of a few points completely changes the character of the anesthetic. This is often evident in young children where a swing from too deep to too light anesthesia occurs very rapidly and with only a slight variation in the mixture of gases. The cry of a saturation may in a few seconds become a reflex cry due to approaching consciousness. For this reason, if parents or friends are present at the operation, they are told that the child may cry or move but that no pain will be felt. They are often unconvinced until they ask the child afterward if he was hurt. This cry, which occurs in a certain percentage of cases, is disconcerting and is one of the disadvantages of nitrous-oxid-oxygen anesthesia for young children.

PRELIMINARY MEDICATION.

There is no anesthetic where the preoperative administration of morphin or other narcotic is more helpful than with nitrous-oxid-oxygen. This is especially so with children, whose demand for oxygen is greater than in adults on account of their increased metabolic rate. The effect of the narcotic is to reduce the rate of metabolism and therefore the demand for oxygen, so that a higher percentage of nitrous-oxid may be given, with a resultant better and smoother anesthetic. However, with the idea of avoiding its depressing effect upon the functions of the body generally, as well as the nausea and vomiting it produces in some patients, the preliminary narcotic was omitted in the majority of cases.

PRELIMINARY VISIT.

If possible, all patients are seen in their rooms before coming to the surgery. A general examination is made and the winning of the patient's confidence is attempted. Not infrequently some condition, such as an acute cold, bronchitis, râles which suggest T. B., increased temperature or acidosis are discovered, which makes a postponement of the operation advisable.

A pleasant induction is especially important for children. Many an adult has postponed an urgent operation until too late because of a disagreeable experience with anesthesia in childhood. If the anesthetic can be so administered that only pleasant memories are associated with it, future anesthetics, if necessary, will not be dreaded. The writer considers this so important that he hopes you will pardon him for going into some detail as to its accomplishment. The confidence of a child can be more easily won in his room than in the surgery. Upon entering the room the patient is greeted by his Christian name and shaken hands with as though he were an adult. After the chest examination he is told that this is to be very much like a party, where everyone dresses in party clothes like the nightgown he has on, that we will play a game and later ice cream will be served.

The game is to see who can go to sleep first with the breeze blowing on the players' faces. He is to hold the "breezer" himself and to blow it first on the anesthetist and then on himself and that it makes a noise like whish-whish-whish. He is told that if he beats the game Dr. So and So is going to take out his tonsils and adenoids when he is asleep and that most boys and girls have a sore throat after. Reference is then made to the ice cream and that a quart or two will be ordered if he wishes it. This takes his mind away from the sore throat but he will realize afterward that he was not deceived. The game of going to sleep is practiced in the patient's room, the child holding the bowl of the stethoscope as a substitute for the "breezer."

When he is placed on the operating table he is given the nasal inhaler to hold, and the great majority of children will go to sleep smiling and repeating "whish-whish-whish." The volume of nitrous-oxid is gradually increased, and the patient

loses consciousness without the inhaler touching the face. Children who come back for a second operation usually look forward to the pleasure of playing the "game."

TECHNIC.

Pillows are placed lengthwise under the patient's body and one small and one large pillow under the head. After the patient is anesthetized the larger pillow is removed so that the head will be extended in order that blood will gravitate away from the lungs. The small pillow gives support to the back of the neck. Further extension or flexion of the head is accomplished by raising or lowering the head of the table. The patient is not strapped down or the mouth gag inserted until after the patient is asleep. If there is difficulty in opening the mouth the anesthetic is lightened, if necessary, to the point of analgesia, when the patient is requested to open the mouth. This is not often necessary, as most jaws can be more or less easily opened. The bowl of a stethoscope with a long tubing is loosely placed over the precordium and the heart action observed during the anesthetic. Should the heart become irregular the mouth gag is loosened and a breath of pure oxygen given, when the irregularity promptly disappears.

Reflected light is used by the surgeons with whom I work. This is better than direct light, as it allows the anesthetist to observe color and the eye reflexes better.

With direct lighting—that is, when the light is attached to the surgeon's forehead—the patient is often temporarily in the dark when the surgeon turns his head. If the anesthetic becomes too light the mouth is either covered with a wet towel for a few seconds or the nasal inhaler is transferred from the nose to the mouth, while the patient's nose is pinched to prevent air dilution through the nasal passages. Should the anesthetic be accidentally pushed during the operation until the patient has stopped breathing, which has happened only a few times in my experience, the mouth gag and other instruments are removed, the face mask substituted for the nasal inhaler and a breath or two of oxygen is forced into the lungs. As the mouth hook is also removed, it is necessary to turn the stopcock at the opposite end of the rubber tubing, for if this were not done it would not be possible to build up sufficient

pressure. The face mask with the expiratory valve tightened is always at hand for this emergency. It is a good plan, when forcing oxygen into the lungs, to have the surgeon exert pressure over the epigastrium to prevent oxygen entering the stomach. These manipulations take but a few seconds of time. Following the suggestion of Dr. Dewitt H. Sherman of Buffalo, a gauze sponge is held under the tonsil before it is removed in order to catch any pus or secretion that may be squeezed out. It is often difficult to keep the lips of the patient from getting between the mouth gag and the teeth. This is especially liable to occur when, after both tonsils are out, sponges are held in the tonsillar fossa, as the sponge holders pull down the lips. At this time it is often difficult in young patients to maintain the desired depth of anesthesia, as the sponges, which are usually too large, interfere with breathing.

Although the nasal inhaler is fastened to the head by the elastic head harness, it is necessary to exert further pressure with the hand in order to prevent a leak between the inhaler and the face. This is especially necessary if there is nasal obstruction and high pressure is required. If the blow prevents the surgeon from locating a bleeding point it can be temporarily stopped. Young children are not fastened to the operating table but are held by a nurse so that they may be quickly turned after the adenoids have been removed. After the operation the patient is given pure oxygen for a few minutes.

Many patients empty their stomachs as they are regaining consciousness, but usually do not recall it afterward. The majority of patients do not need to be lifted from the table to the cart, as they are fully conscious and can move themselves.

AFTER-CARE.

Adult patients, and occasionally children, are given aspirin to be dissolved on the tongue every four hours for the first day or two, to lessen the pain of swallowing.

Soft food, such as ice cream and custards, is given a few hours after operation if the patient desires it. Children usually leave the hospital the same day, if operated upon in the morning, and adults usually the day following.

The cost of the nitrous oxid and oxygen varies with different patients. Mouth breathers require more than twice the

volume that nose breathers use. The average cost per hour when the large cylinders are used is about ten dollars.

CONCLUSIONS.

The appetite usually returns more quickly and there is less nausea and vomiting when no ether is used.

The technic of doing tonsillectomies under nitrous-oxid-oxygen is worth developing, if only for those cases where ether is contraindicated.

For children under three years of age, unless there is a contraindication, it is usually better to add a trace of ether and give a higher percentage of oxygen in order to avoid jactitation.

In this series of cases there have been no deaths and no lung abscesses.

519 FRANKLIN ST.

X.

COLONIC ANESTHESIA IN OPERATIONS OF OTOLARYNGOLOGIC CASES.

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The history of the use of a mixture of ether in oil as an anesthetic agent dates back for a considerable time. Nevertheless, it has only been within the past few years that this agent when injected into the colon has proven satisfactory to surgeons as well as safe to the patient in the production of surgical anesthesia.

Like other special methods in common use, colonic ether is one that is best used in selected cases, and very satisfactory results may be obtained, but only when a more or less standard technic is employed.

Colonic oil-ether anesthesia is particularly adaptable to surgical procedures in and about the head and neck, and especially in those so-called major ones, namely, extensive sinuses, prolonged and difficult mouth operation, laryngectomy, brain surgery and plastic surgery of the face.

As to routine tonsillectomy and simple mastoid, this method is rather a formidable affair.

Rectal anesthesia has its indications and contraindications as well as all other forms. Of these probably the greatest is the poor condition of the patient. In those so-called "poor surgical risk" cases, rectal anesthesia should be used only when other methods are not possible, and then only when an operation is imperative.

By this method alone it is in a certain large percentage of cases difficult to obtain deep surgical relaxation, but as deep anesthesia is not often desired in types of cases in which rectal anesthesia is mostly used, this fact can hardly be called a contraindication for its use.

Its use is certainly to be forbidden in cases where there is the presence of pathology in the rectum, as hemorrhoids, ulcers,

fistulas and colitis. Such conditions cause so much disturbance to the patient in the administration, and the after-effects prove so troublesome from the local irritation of the ether that some other method must be chosen.

Again, because of the necessity of the rather lengthy preparation of the patient, it is impracticable in emergency work.

Last, but not least, there is a certain element of lack of control on the part of the anesthetist of his agent. It is true one can unclamp the tube, syphon out the remaining mixture in the colon and then wash it out, but this takes time, and in those patients that suddenly go into deep anesthesia or shock, every minute counts.

The advantages are very definite. By this method the anesthetist is not in the field of operation, and even though he is surgically prepared, the absence of another pair of hands proves advantageous to the surgeon as well as insuring a better aseptic technic.

No matter what the preliminary medication may be, there is very often a degree of apprehension on the part of the patients. With the colonic type of anesthesia this is lessened. They do not object to an enema, and, if carefully given, no discomfort is manifested by them, the patients going quietly to sleep in a natural manner.

In the absorption of the gaseous ether by the membrane of the intestinal tract, as it separates from the oil, it passes through a greater part of the whole circulation and thereby becomes warmed to body temperature. This causes less irritation to the lungs and air passages, as the ether vapors are given off from the lungs and thereby lessens the danger of postoperative lung complications.

The after-effects, especially nausea and vomiting, are greatly lessened. In other forms of open administration of ether a large amount of mucus and saliva become saturated with ether and certain amounts enter the stomach. This is avoided in colonic administration.

While the number of operations performed with this method of anesthetization to date is not very large, those using it have noted very few accidents occurring, also the low mortality. This is probably due to the fact that the rate of absorption is rather constant, being about two ounces per hour.

There are several rules or principles of administration which must be adhered to rather rigidly:

First.—The apparently simple manner of injection and the ease with which it is done may lead to carelessness, and so watchfulness at all times is a doubly necessary precaution.

Second.—There is a tendency to follow the same procedure with its dosage, etc., in every case of the same type. Each case must have careful study as to time of operation, percentage of drugs used and the general physical condition of each individual patient.

The preparation should, whenever possible, begin the second night before operation with a laxative, the one of choice being castor oil. The practice of giving a laxative the night before the operation, even though mild, should be avoided, especially in those cases scheduled for early operation; for such a routine may cause purgation about the time the colon should be quiet and cause difficulty in the retention of the oil-ether mixture.

If success is to be obtained there must be a thoroughly clean rectum and lower colon. This is best obtained by high colonic irrigations with soapsuds enemas, the first given twenty-four hours before, the second twelve hours, and the possible third at least two hours before beginning the anesthesia.

All of these flushings should be given by a nurse and in sufficient quantity until they are returned clear.

The night before the operation a good night's sleep should be insured by giving some sedative, such as morphin, gr. ¼, or luminol or other hypnotic at bedtime.

The diet of the previous day should be light in character, thereby limiting the bulk of the intestinal contents.

The administration should be undertaken only by one familiar with the different stages to be encountered and with a thorough knowledge of ether. The percentage to be used varies within certain limits. Gwathmey's rule of one ounce of a 75 per cent solution to each twenty pounds of body weight (but never more than eight ounces) is surely safe and satisfactory to follow. In a child the percentage should be nearer one of equal parts, but, as in all anesthetics, the general picture of that individual case must be considered.

The injections are best given in the patient's room, and the less disturbance, the quieter the patient and the more satis-

factory the effect. The necessary paraphernalia should be handy, and consists of a rectal tube, about No. 24, with a clamp attached, a funnel or Murphy drip bottle, an airway and tongue forceps and a freshly prepared mixture of ether and oil which has been warmed. The actual administration consists of three parts:

First.—One and a half hours before the operation a preliminary enema is given of equal parts of oil and ether, the amount varying from one to four ounces, depending upon the physical development of the patient and the depth of anesthesia desired. The writer has found that an ounce, or even two of each ingredient, while in itself not sufficient to produce anesthesia, gives an analgesia which increases the efficiency of the second or larger enema to follow.

To this first injection is added ten grains of chlorentone in solution or by suppository, the drug acting as a local anesthetic to the membrane and causing less disturbance to the patient.

Second.—An hour before operation a sixth or one-quarter grain of morphin sulphate with one-hundredth grain of atropin is given hypodermatically.

Third.—This procedure is to start about 30 or 40 minutes before operation and because of the previous enema and medication should be carefully observed. The patient is placed in the left Simms position, whenever it is possible, with a pillow under the left hip, allowing the mixture to be spread by gravity over a larger area of colon for better absorption.

The rectal tube is inserted into the rectum about four to six inches, and the warmed mixture of 60 to 75 per cent oil-ether is allowed very slowly to run into the colon, taking at least ten minutes to complete the injection. If there is hurry at this time, the patient complains of pain and has a strong desire to expel the contents.

A little firm pressure over the anal opening will generally prevent any such accident. The tube should be allowed to remain in the rectum and should be clamped.

To this mixture, before administration, may be added a dram of paraldehyde. This aids in producing a more even sleep and greater relaxation with less absorption of ether.

In about ten to fifteen minutes the ether may be easily detected on the patient's breath, and he shortly goes through the

well known stages of ether anesthesia. As a rule, there is less of the excitement stage, and the narcosis is a smooth, easy anesthetization.

It should always be borne in mind that the early stages of colonic ether anesthesia are probably the most dangerous, for here it is that the idiosyncrasies of the patient show themselves.

The danger signs are as follows:

1. The patient may succumb to the anesthetic during the administration, and if this does occur the procedure must be stopped at once.
2. A rapid diminution of reflexes.
3. Cyanosis, even though slight.
4. Embarrassed respiration, even though slight.

If the patient becomes cyanotic, the cause must be determined, if possible. It may be due to relaxation of the jaw and tongue or obstruction to the air passages by mucus or saliva. If these causes are eliminated by bringing the tongue forward and using the airway and there is still cyanosis, the solution must be withdrawn, and means taken to combat the probable depression of the respiratory center.

In a certain number of cases the slow absorption is such that surgical anesthesia is delayed. This may be hastened by cutting down the escape of ether from the lungs by placing a wet towel or gauze over the mouth, or by supplementing with inhalation anesthesia for a few minutes.

These methods are far safer than adding more mixture to the rectum. Generally when relaxation is obtained the anesthetic may be withdrawn and the anesthezation goes on in a very satisfactory manner.

All during the course of the operation just as much care must be exercised as in any other method. The signs and symptoms are like those of inhalation, or any other type, and the depth, etc., may be observed equally as well.

After the operation is finished or nearly so, the remainder of the mixture should be syphoned off and the bowel thoroughly irrigated with warm saline solution. If there is too deep an anesthesia, or if shock is present, the injection of a pint of strong black coffee, which is allowed to remain, will hasten the recovery.

As a rule, patients in whom this form of ether anesthesia is used are slow in regaining consciousness, and if so the administration of oxygen, or, better, a carbon dioxid mixture, will hurry the recovery.

CONCLUSION.

Ether-oil method of anesthesia is a safe procedure when carefully carried out and is particularly adaptable to major work in nose and throat cases. Care must be exercised at all times, as in all forms there must be cooperation between the surgeon and anesthetist. These are certain definite rules of procedure which must be observed if success is to be assured.

The entire procedure must be under the direction of a person who is properly trained in such management.

5052 WALNUT ST.

XI.

RESPONSE OF VESTIBULAR APPARATUS TO DRUGS.*

By Ellison L. Ross, M. D., and Murland W. Fish, M. D.,

Otolaryngologists have long felt more or less helpless in relieving vertigo originating in the vestibular organ. A considerable number of drugs have been employed by various men at various times. None have been effective to any satisfactory degree.

Jonkhoff[1] administered small and large doses of strychnin to animals. He found that small doses increased all labyrinthine reflexes. Larger doses caused a disappearance of various phases of vestibular response.

Jonkhoff[2] used picrotoxin in small doses and produced a distinct and remarkably prolonged increase in labyrinthine reflexes. Larger doses brought on a decrease of the same reflexes.

The same author,[3] in working with rabbits and administering camphor, produced a stimulation of the reflexes with small doses and a depression with large doses.

Kleyn and Versteegh[4] have devised a method which is intended to show the action of a drug on the various parts of the peripheral and central vestibular apparatus. They used nicotin on rabbits. They concluded that the drug acted peripherally on the ocular muscles to contract them. Nicotin stimulated the vestibular nucleus to shorten the rapid phase of nystagmus.

Versteegh[5] administered alcohol to rabbits. He observed the gradual loss of postural reflexes with the increasing doses. Those of the head were the last to disappear.

Bijlsma and Versteegh[6] administered chininon and hydrochininon to mice and guinea pigs. Intoxicating doses produced

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rolling movements much like those of animals with the destruction of the vestibular apparatus. As proof of its not being due to action on the vestibular apparatus, it was found to be unaffected by destruction of one or both labyrinths.

Jonkhoff[7] administered oil of chenopodium to guinea pigs and rabbits. He observed a stimulation of labyrinthine reflexes.

Pearcy and Hayden[8] reported that 3 to 5 grains of sodium nitrite every two hours relieved seasick persons of nystagmus, vertigo, ataxia and nausea within four hours.

There are a number of drugs that have fairly clear-cut specific actions. Epinephrin stimulates the endings of the nerves of the thoracolumbar sympathetic nervous system. Blood pressure is raised by it. Heart rate is increased by it. Atropin deadens the endings of the nerves of the craniosacral sympathetic system. The chief nerves of this system are the vagus, third nerve and the chorda tympani. Atropin, through its action on the vagus, increases the heart rate, dilates the bronchioles and reduces peristalsis. Through its action on the third nerve the pupil is dilated and the accommodation is paralyzed. Pilocarpin stimulates where atropin depresses. Many more such examples of specific drug action come to mind.

If the vestibular nerve could be accurately placed in our classification of the nervous system, it would be a simpler problem to find a drug that would stimulate it and one that would depress it. Is it purely a sensory nerve? Does it belong to the thoracolumbar sympathetic system which has to do with increasing the heart rate and the raising of blood pressure? Does it belong to the craniosacral sympathetic system which includes the third nerve, vagus and chorda tympani?

The physiology of the vestibular apparatus furnishes some basis for the classification of the nerve.

Through the action of light on the retina we have vision. Through the action of the vibration of air on the drum and various parts of the ear we have hearing. Through the action of gravity and the fluids of the semicircular canals on the specialized nerve endings in the vestibular apparatus we have a sense of movement in relation to the direction of gravity. Viewing the subject from this angle, we must consider the vestibular nerve one of special sense, like the optic, auditory

and olfactory nerves. This view is held by various physiologists.

While the vestibular nerve, as far as function is concerned, is one of special sense, it does not possess all of the qualifications found in other members of the same group. Vision gives quantitative knowledge of color, form, size, distance, movement, etc. The vestibular apparatus gives us only qualitative knowledge of head movements.

While there are a few drugs that do affect the senses temporarily, it is possible that the rather "unorthodox" vestibular sense might be specially acted upon. Among the cranial nerves, numbers 1, 2, 5, 7 and 8 have special sense functions. Nerves 5, 7, 9 and 10 have sense functions. Nerves 3, 4, 5, 6, 9, 10, 11 and 12 have motor functions. Nerves number 3, 7 and 10 are partially or completely acted upon specifically by the atropin-pilocarpin group of drugs and belong to the craniosacral sympathetic nervous system. This heterogeneous distribution of functions and susceptibilities to drug action among the cranial nerves makes conclusions from deductions absolutely impossible. Therefore it is proposed to make a series of tests of the action of drugs on the vestibular function.

EXPERIMENTAL WORK.

In order that our results might be most directly applicable to our medical practice, mutilating operations, employment of lower forms of animal life and unphysiologic methods of stimulation were avoided as much as possible. Dogs were used as subjects. No operations were performed. The method of stimulation was rotation, with the dog's nose in the vertical plane. The duration of nystagmus was taken as the measure of the degree of stimulation.

The dog was tightly tied to a specially constructed board so that no part of the body could change position. A large leather muzzle, fitted with a 20 diopter lens, was placed on the dog's head in such a way that one eye was covered with leather and the other with the lens.

The board was then attached to a vertical bar which by means of an electric motor was rotated at a constant rate of about 35 turns per minute. From a series of tests we decided that rotation for fifteen seconds with a sudden stop produced

a vestibular stimulation which was not maximum and still large enough to allow a considerable diminution. After the sudden stoppage of the dose of rotation, the duration of nystagmus was timed with a stop watch. The interval between tests was always five minutes. Usually three tests were made before the drug was administered. The drug was always injected intraperitoneally. Two minutes after the drug was given the next test of rotation was made. The tests were continued for about an hour after administration of the drug.

RESULTS.

A series of ten dogs that had not been given any drug was run through the tests to determine the limit of normal irritation. Table No. I contains the data collected on this series.

It is to be noted from this table that normal dogs varied considerably as to the sensitiveness of their vestibular apparatus. Some dogs were very much more sensitive than others. Each dog varied considerably in his response to the various tests of the series. From the averages, we find that the rule was to start with about 22 seconds of nystagmus and gradually decrease to about 18 seconds after seven tests. The following seven tests remained between 18 and 19 seconds.

The next series of dogs was tested after the administration of epinephrin. After the first three tests each dog was given 0.1 cc. of 0.1 per cent epinephrin solution per kilogram of body weight. The results are given in Table No. II.

Several of the animals made decided increases after the drug. Dogs 18, 19, 20, 50, 51 and 54 made decided increases in the duration of nystagmus over the last observations before the drug was given. However, in the group of normal dogs an equal number showed decided increases at some time after the third test. Dogs 1, 2, 4, 6, 7 and 9 showed decided increases over the third test. Out of the series of dogs given epinephrin only 40 per cent showed a decided increase over the longest period of nystagmus before the administration of the drug. Dogs 18, 20, 21 and 54 increased their periods of nystagmus after the administration of the drug decidedly above the longest of the first three observations. Of the normal group of animals 30 per cent increased their periods of nystagmus above any of the first three tests. Dogs 2, 8 and 10 had periods of

nystagmus after the first three tests greater than any of the first three. If the averages of the normal group and the epinephin group are graphed, it will be seen that the curves are very closely parallel to each other. Therefore it must be concluded that epinephin has not altered the sensitiveness of the vestibular apparatus.

Nitrites have been proposed for the purpose of reducing vertigo. Therefore a series of seven dogs was tested. The results are found in Table No. III. Each animal was given a large dose, varying from .5 to 1.0 cc. of 5 per cent solution of sodium nitrite per kilogram of body weight. In all cases the systemic reaction was marked. In every case there was a decided drop in the period of nystagmus. This occurred in all animals between the second and fourth tests after the drug. The averages show the first decided fall to be in the fourth test after the drug. There was also a late fall in most cases due to impending death. The third and fourth dogs died at the end of the series. It must be concluded that large doses of sodium nitrite did depress the vestibular response to stimulation.

Cocain was used on a series of six dogs. A dose of 10 mg. of cocain hydrochlorid per kilogram of body weight was used. There were decided systemic effects, such as tremors and excitement. The results are given in Table No. IV. In this group of animals there was a decided and general decline in reaction to stimulation. This is shown individually and in the averages.

Nicotin was used on six dogs. The dose employed was 0.5 mg. per kilogram of body weight. The general effect evidenced was excitement. The results are tabulated in Table No. V. It is striking that these animals began on quite different scales. However, the degree of change was approximately that of normal animals. In the comparison of the normal average series with the average series of this group of animals they are found to be quite closely parallel to each other. There is no evidence that nicotin had any decided effect on the vestibular response to stimulation.

Pilocarpin, a stimulator of the endings of the vagus, chorda tympani and the third nerve, was used on a group of five dogs. The dose was large, consisting of one-third of a cubic centimeter of 1 per cent pilocarpin per kilogram of body weight. The results are tabulated in Table No. VI. There was prostration

and increased peristalsis in every animal of this series. In no case was there an increase after the drug was given. The averages show a decided depression of the vestibular reaction due to the drug.

Atropin, which depresses where pilocarpin stimulates, was used on a group of animals. A heavy dose of 1 cc. of 0.1 per cent atropin sulphate solution per kilogram of body weight was used. The results are found in Table No. VI. There was excitement and pupillary dilation in all cases. The individual variations were within normal limits. Group averages compare favorably with the averages of normal animals. This group of animals showed no effect of atropin on vestibular reaction to stimulation.

DISCUSSION AND CONCLUSIONS.

Six drugs have been used on animals to detemine their effects on the response of the vestibular apparatus to stimulation. Three of them, sodium nitrite, cocain and pilocarpin, have decreased it, and three, epinephrin, nicotin and atropin, have had no effect.

All of the drugs that decreased vestibular response had severe depressive reactions on the animals in general. The doses were maximum and several animals died shortly after the observations. *The actions of these three drugs differ widely. Nitrites relax the smooth muscle fibers of the body. Cocain stimulates the endings of the thoracolumbar sympathetic nerves. Pilocarpin stimulates the endings of the craniosacral sympathetic nerves. Therefore we are forced to conclude that these drugs had no specific effect and the only reason for the decreased reaction was the general depression due to prostrating doses of the drugs.

With no effect on vestibular reaction through the action of epinephrin, nicotin and atropin, a number of deductions can be made. Both epinephrin, and to a certain extent, nicotin, stimulate the nerve endings of the thoracolumbar sympathetic system. If any of the nystagmus and vestibular mechanism were governed by the thoracolumbar system, we would surely

*Epinephrin was first reported as increasing vestibular response, but more data included in this paper prove it otherwise.

have gotten some alteration by the action of epinephrin or nicotin. Since the dose of nicotin was sufficient also to paralyze the thoracolumbar sympathetic ganglions, we are forced to conclude that they have nothing to do with the nystagmus response from vestibular stimulation.

Atropin paralyzes the endings of the craniosacral sympathetic nerves. Since this drug did not have any effect on the response to labyrinthine stimulation, we must conclude that the nerves involved in the nystagmus response to vestibular stimulation do not belong to the craniosacral sympathetic system.

Further work on this subject is being conducted by the authors.

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TABLE NO. I.

DURATION OF TEST-NYSTAGMUS IN NORMAL DOGS.

Results expressed in seconds.

Test No.	Dog 1	Dog 2	Dog 3	Dog 4	Dog 5	Dog 6	Dog 7	Dog 8	Dog 9	Dog 10	Average.
1	26.0	21.6	23.0	21.8	24.8	21.6	24.0	17.2	20.4	23.2	22.4
2	23.8	22.0	19.0	18.0	22.2	22.6	21.8	17.6	19.4	24.0	21.0
3	20.6	20.4	16.0	16.0	23.0	22.0	19.0	19.0	18.2	24.2	19.8
4	21.8	20.0	16.4	16.4	21.8	21.8	20.2	18.4	20.2	21.8	20.0
5	20.2	19.8	15.0	16.8	20.6	21.8	19.4	18.6	18.2	23.2	19.4
6	19.4	19.6	15.0	17.0	19.4	22.0	18.2	19.0	17.8	25.4	19.3
7	19.2	20.0	.....	17.4	19.0	22.8	18.0	18.4	18.4	22.6	19.5
8	19.0	19.6	14.8	18.0	18.8	19.8	17.4	19.6	17.6	22.6	18.7
9	19.2	20.2	13.8	16.0	20.0	25.0	16.2	18.2	18.0	21.8	18.8
10	19.4	21.6	12.8	17.6	17.0	22.2	16.6	19.0	17.4	22.4	18.6
11	18.8	21.6	13.0	17.2	18.0	20.2	16.6	18.8	18.4	22.6	18.5
12	19.0	22.6	14.8	.....	17.4	19.8	15.8	19.0	17.0	.....	18.2
13	19.6	22.0	13.4	16.6	17.4	21.6	16.0	.....	17.6	22.0	18.5
14	18.8	22.6	12.8	16.4	16.6	19.8	17.2	.....	17.4	22.0	18.2

TABLE NO. II.

DURATION OF TEST-NYSTAGMUS IN DOGS WITH EPINEPHRIN.

Results expressed in seconds.

Test No.	Dog 18	Dog 19	Dog 20	Dog 21	Dog 50	Dog 51	Dog 52	Dog 53	Dog 54	Dog 55	Average.
1	17.8	22.6	23.0	18.2	15.8	22.2	19.6	17.2	19.4	21.8	19.8
2	17.0	19.0	21.6	18.0	14.6	22.6	18.2	16.2	18.2	21.0	18.6
3	16.8	17.2	21.6	20.0	14.0	18.8	15.2	14.6	19.2	20.0	17.7
Injected 0.1 cc. of 0.1 per cent epinephrin per kilogram.											
4	18.0	19.0	24.2	16.0	14.4	18.0	15.0	14.0	17.4	19.2	17.5
5	19.8	19.0	25.6	14.2	15.6	17.8	14.2	14.8	15.8	20.2	17.7
6	19.4	19.6	26.2	13.0	14.0	18.0	13.8	14.0	17.8	19.4	17.5
7	18.4	18.0	23.2	15.8	13.0	17.0	14.4	13.4	21.8	17.8	17.3
8	17.8	16.4	21.8	18.0	14.2	19.4	13.2	13.2	21.4	16.4	17.2
9	17.8	17.0	20.8	21.0	12.2	19.0	14.4	14.0	18.8	16.2	17.1
10	17.6	17.2	20.8	18.0	.....	18.0	14.2	13.6	17.2	17.2	17.1

TABLE NO. III.

DURATION OF TEST-NYSTAGMUS IN DOGS WITH SODIUM NITRITE.

Results expressed in seconds.

Test No.	Dog 11	Dog 12	Dog 13	Dog 14	Dog 15	Dog 16	Dog 17	Average
1	21.8	27.2	22.2	21.2	24.8	17.6	.....	22.5
2	18.8	21.2	20.2	17.6	21.8	16.0	12.0	18.2
3	19.2	20.4	19.4	16.6	20.4	15.0	12.2	17.6
Injected sodium nitrite 0.5 to 1.0 cc. of 5 per cent per kilogram.								
4	18.6	17.4	18.8	15.8	19.8	14.6	11.0	16.6
5	16.0	17.6	15.0	15.0	18.8	14.2	12.0	15.5
6	18.2	16.8	15.4	15.0	19.6	13.2	12.6	15.8
7	17.0	16.6	15.6	15.2	19.4	13.4	9.8	15.3
8	14.2	17.4	23.4	15.2	19.6	12.0	10.2	16.0
9	14.4	17.0	21.8	15.4	19.0	11.6	10.0	15.6
10	12.6	16.2	Dead	16.2	18.6	14.4	9.0	14.5
11	.....	15.8	.....	16.0	18.6	11.4	10.2	14.4

TABLE No. IV.

DURATION OF TEST-NYSTAGMUS IN DOGS WITH COCAIN.

Results expressed in seconds.

Test No.	Dog 25	Dog 26	Dog 27	Dog 29	Dog 30	Dog 31	Average
1	24.2	21.8	18.8	17.0	19.6	26.6	21.3
2	22.2	19.4	15.6	16.0	19.0	23.4	19.3
3	18.4	19.6	15.6	.....	.....	23.6	19.3
Injected 10 mg. cocain-HCl per kilogram.							
4	17.8	18.2	14.0	16.0	18.0	23.0	17.8
5	17.4	19.2	13.2	13.0	15.6	19.0	16.2
6	15.8	21.0	11.8	12.6	15.8	18.2	15.9
7	16.0	17.6	12.8	11.6	14.0	13.6	14.3
8	14.8	17.2	12.0	11.0	14.6	.....	13.9
9	14.8	16.8	10.6	13.0	15.0	14.6	14.1
10	14.4	15.4	12.0	12.6	14.2	.....	13.7
11	14.4	16.4	.....	12.0	15.0	15.0	14.6

TABLE No. V.

DURATION OF TEST-NYSTAGMUS IN DOGS WITH NICOTIN.

Results expressed in seconds.

Test No.	Dog 32	Dog 33	Dog 34	Dog 35	Dog 36	Dog 37	Average
1	15.4	19.4	22.2	16.6	23.0	28.8	20.9
2	12.4	17.6	22.0	15.6	24.4	22.2	19.0
3	12.0	17.4	21.6	15.4	22.8	23.2	18.7
Injected 0.5 nicotin per kilogram.							
4	11.6	17.8	22.4	16.2	23.0	19.0	18.3
5	12.8	17.0	23.6	13.8	23.0	19.2	18.2
6	12.4	16.6	21.4	13.6	22.6	19.6	17.7
7	11.0	16.0	19.0	13.4	23.4	.....	16.6
8	11.6	15.8	18.8	13.8	23.6	21.0	17.4
9	11.4	15.0	18.4	13.8	23.0	20.4	17.0
10	11.0	15.0	19.4	14.4	.....	20.0	16.0

TABLE No. VI.

DURATION OF TEST-NYSTAGMUS IN DOGS WITH PILOCARPIN.

Results expressed in seconds.

Test No.	Dog 38	Dog 39	Dog 40	Dog 41	Dog 42	Average
1	21.0	21.6	19.8	17.2	25.4	21.0
2	20.4	22.0	14.4	18.6	22.0	19.5
3	19.8	17.4	15.0	18.6	22.0	18.6
Injected 1 cc. of 1 per cent solution pilocarpin per kilogram.						
4	19.8	16.8	10.0	18.6	19.0	16.8
5	18.4	15.0	10.0	17.0	20.0	16.1
6	18.8	13.4	9.6	11.8	20.0	14.7
7	19.6	.....	6.6	10.0	13.6	12.5
8	17.0	15.0	7.0	12.3	18.0	13.9
9	18.4	16.4	.....	9.3	18.0	15.5
10	17.8	17.6	.....	11.3	15.0	15.4

TABLE No. VII.

DURATION OF TEST-NYSTAGMUS IN DOGS WITH ATROPIN.

Results expressed in seconds.

Test No.	Dog 43	Dog 44	Dog 45	Dog 46	Dog 47	Dog 48	Average
1	22.6	19.4	19.8	27.2	22.8	29.4	23.5
2	18.8	18.0	20.2	22.4	23.6	19.6	20.4
3	17.8	17.4	20.0	18.6	20.8	19.6	19.0
Injected 1 mg. atropin sulphate per kilogram.							
4	17.4	15.0	19.0	19.2	21.4	20.0	18.7
5	18.8	14.4	17.4	19.4	24.2	18.6	18.8
6	17.6	15.0	19.0	15.4	21.2	18.2	17.7
7	17.8	16.0	20.0	15.2	23.6	18.4	18.5
8	17.0	15.6	19.4	16.4	23.4	20.0	18.6
9	14.8	.....	19.2	16.6	22.0	19.4	18.4
10	.....	17.2	19.0	15.4	20.0	19.0	18.1

XII.

OTITIS MEDIA AND ALLERGY.

EUGENE R. LEWIS, M. D.,

LOS ANGELES.

I have encountered six cases of acute otitis media during the past decade which attracted my attention by strongly suggesting allergic nature of the inflammatory reaction. Unfortunately I was able to elicit something definite in the history of only two cases respecting allergic troubles in progenitors.

Male, age 45, son of allergic (asthmatic) mother, father of an allergic (asthmatic) son; attended dinner party where shortly after being seated at the table he began to notice an odd sensation in left ear. This quickly became an increasing earache, throbbing and obtunding hearing, and before the close of the meal spontaneous perforation with profuse serous discharge had taken place. Examination prior to this incident had revealed intact tympanic membrane and quiescent tympanic and eustachian structures. Temperature, pulse and respiration were normal, and upper respiratory tract showed no symptoms or evidence of inflammation that evening or the following days. The serous discharge was profuse for the first fifteen hours; the patient was kept in bed, given a high fluid intake with alkalizing citrates and bicarbonates. In thirty-two hours the discharge ceased and eustachian inflation showed perforation closed the next day.

Female, age 42, able to give very little information concerning health of progenitors; while shopping one morning experienced acute right earache, without premonitory symptoms of any kind. Examination showed tympanic membrane swollen and pink, and spontaneous perforation occurred during otoscopic inspection, about ninety minutes after onset of symptoms, with abundant clear serous discharge. She was put to bed at once; temperature, pulse and white cell count were normal; cochlear examination showed high degree of sound conduction impairment; general upper respiratory examination discovered no evidence of irritation except closure of right

eustachian tube. Heat applied to right ear, high alkaline fluid intake and rest in bed for thirty-six hours resulted in abrupt cessation of discharge, subsidence of tympanic swelling, opening of eustachian tube and closure of perforation within thirty-six hours of cessation of serous discharge. This patient subsequently had a similar experience in San Francisco under otologic observation, details of which were sent me by her otologist.

Four other cases, two of which were children, 7 and 9 years old, respectively, and two adults—were seen by me recently; each showed acute earache, marked sound conduction impairment, very early spontaneous serous discharge which ceased completely in from thirty-six to seventy-two hours with prompt closure of perforation and restoration of hearing. The history of the 9-year-old boy indicated definite family history of allergy (eczema, hives and hay fever) in one parent. In none of these cases was the nature of the allergen apparent; absence of leukocytosis suggests noninfectious nature of the acute inflammatory process, which is further borne out by the clinical course of each case.

Regarding allergy in general, the following summary seems reasonably well established: "Abnormal local tissue reactions" may be subdidived into:

1. Allergy—always inherited; protein etiology.
2. Anaphylaxis—always acquired; protein etiology.
3. Altered reactivity—always acquired; nonprotein etiology.

Regarding the tissue cell itself involved in abnormal local tissue reactions it is conceivably due to conditions within cytoplasm, conditions outside cytoplasm, or both. Allergic cell conditions are definitely permanent, and considerable evidence points to probability of intracellular locus of the determinant condition or conditions; this is not so evidently probable respecting anaphylaxis, which is not a permanent condition; nor is it probable respecting altered reactivity, which also is not a permanent condition.

Regarding the mechanism of abnormal local tissue reactions, all of them—both protein and nonprotein—involve body surfaces in the contacts which culminate in reactions, the skin, mucous membranes and serous membranes. Pseudopods are

characteristic of observable local reactions to protein; they are not seen in nonprotein reactions, such as are elicited by quinin, aspirin, salvarsan and similar agencies.

Regarding incidence in individuals, allergy occurs according to Mendel's law; anaphylaxis and altered reactivity do not. In brief, mating of dominants, recessives and hybrids results with respect to allergic offspring as follows:

Dominant with recessive—no allergy; offspring hybrid.

Dominant with hybrid—no allergy; offspring hybrid.

Recessive with recessive—allergy; offspring recessive.

Recessive with hybrid—allergy; offspring hybrid.

Hybrid with hybrid—allergy; offspring hybrid.

This shows how allergic offspring may be produced by individuals with negative histories of allergy.

Regarding multiple sensitivities, the allergic individual is in all probability originally nonspecifically multiple sensitive; with the passage of time and the consequent increase in the number and variety of protein contacts, an increasing number of specific reactions become manifest. But it would be erroneous to deduce that until each was manifested no abnormal tissue reactivity had existed for the specific allergen in question.

Regarding the general nature of abnormal tissue reactions there are two main avenues of approach for observing the general nature of all these reactions: (1) the study of influences producing the special reactions, (2) the study of influences altering the special reactions. It has been generally accepted that, whatever the condition may be which results in the existence of abnormal tissue reactions, the locus of the condition is intracellular. Inasmuch as there is in the picture, nevertheless, the possibility of extracellular locus, it seems safer to maintain an inconclusive attitude toward this phase of the question. The Russian experimenters bled sensitized rats to the point of practically total ablation of their blood tissues, substituting Ringer's solution by perfusion, after which they found the animals still reacting positively to their sensitizations. From this they deduced that the location of the sensitizations was intracellular. It is manifestly impossible to abstract completely all fluid tissues in such experiment animals; and even granting that possibility there is no reason to believe that the

perfused substitute fluid might not continue to intermediate, as its predecessor may have done, between the reacting cells and a source of allergenesis in some of the body tissues. With the foregoing reservations in mind, it still seems probable that the peculiar condition responsible for these tissue reactions is actually within the cytoplasm of the reacting cells; and, further, that it is self-perpetuating under changing metabolic conditions. Considering Slye's deductions from studies of such inheritances in rodents, that characteristics of inherited tissue tendencies have been laid down in the original protoplasm, we are confronted with a phylogenetic explanation of allergy. The writer has found it difficult to maintain a disbelief in the inheritance of acquired characteristics, despite the inability of scientists to prove such inheritance, or the reverse. In confronting the prevalence of blondes among Nordic peoples, the absence of pigment in arctic animals, such as polar bears, foxes, ermines—and the prevalence of brunettes in tropical peoples, the presence of pigment in tropical animals, one is compelled to choose between two alternatives—belief in inheritance of acquired characteristics, or elimination of brunettes in the north, of blondes in the south, by the operation of the law of the survival of the fittest.

The study of influences altering these abnormal tissue reactions brings us at once to three outstanding agencies—adrenalin, iodin and ephedrin. These may operate by biophysical changes induced extracellularly, by biochemical changes produced intracellularly, or extracellularly, or by activating or inciting something within the organism, such as endocrine potentialities. What is known regarding adrenalin and iodin is strongly suggestive of some sort of endocrine participation in the alterations of allergic reactions observed after the use of adrenalin and iodin.

There are certain characteristics of so-called "acute serous" otitis media which suggest the absence of infectious etiology; and, even more strongly, suggest allergic etiology. It seems probable, also, that many cases of acute otitis media originate as allergic and subsequently become infectious as the result of the operation of secondary bacterial activities.

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XIII.

VERTIGO AS A SYMPTOM.*

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Vertigo as a symptom is of interest to every physician. It occurs in the symptomatology of a vast number of diseases. It varies in intensity but often is of such a degree as to be not only terrifying but incapacitating. The victim demands relief. The physician has a great mystery to solve.

We perhaps should first inquire just what is meant by vertigo. The average patient finds it difficult to describe accurately his symptoms. A feeling of faintness or nausea, a transient hemianopsia or any nerve upset may be called vertigo. But etymologically and in the sense generally accepted by modern writers, and for the purposes of this discussion, vertigo of a rotatory type is meant—that is, the patient experiences a sensation of turning, applied either to himself or to objects about him.

The next problem is to locate the cause. If there are associated symptoms, these may help to solve the problem. If there are none, the mystery is greater.

The causes of vertigo may be divided as follows:

1. Eye disorders.
2. Ear disease.
3. Intracranial disease.
4. Remote disorders and systemic and metabolic disturbances affecting the labyrinth of the ear.

I may be criticised, but I think there is a true ocular vertigo which it is our duty to recognize. If a patient with paralysis of an extraocular muscle looks in the direction of the paralyzed muscle there is an apparent rapid movement of objects in the opposite direction, which constitutes in the meaning of the

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word and in the sensation of the patient a true vertigo.[1] However, if the eyes are closed the symptom at once disappears. Thus we eliminate in a simple manner other causes. For this reason, I think, unless there is obvious ear disease, the eye examination should come first. At the same time the ophthalmoscope should be used to determine if a choked disc or retinal or other lesions are present which might throw some light on the etiology of the vertigo, if the eye muscles are not at fault.

Since the day Flourens[2] published his observations in 1842, the labyrinth of the ear has been recognized as the chief organ controlling somatic equilibrium. Waves of sensation, subconscious in part at least, are constantly proceeding from the semicircular canals and the vestibule, through the vestibular nerve to nuclei in the bulb, whence some pass through connecting neurons to the cortex of the cerebrum, and others through the principal nuclei in the bulb and Deiter's nucleus, through the cerebellum into the motor cells of the anterior horns of the cord. When these sensations originate, and are distributed in all these directions, in a normal manner, their subconscious interpretation results in equilibrium or a sense of equilibrium. But when the impulses at their origin on one side in the semicircular canals and the vestibule are unduly stimulated or retarded, at once the sense of equilibrium is upset and vertigo results. If one labyrinth is stimulated or paralyzed while the other remains normal, vertigo results. Labyrinthine vertigo is always rotatory. Changes of the pressure of the intralabyrinthine fluid and in the circulation of the blood in the labyrinthine vessels irritate the nerve cells of the cristæ and ampullæ, causing vertigo. It is readily understood how very many disorders of the tympanum and tympanic membrane, and even of the external auditory meatus, can bring about these changes, and how disease of the temporal bone may cause serous and suppurative labyrinthitis.

Gross disease of the labyrinth can also occur without preexisting disease of any other part of the ear, as in Ménière's disease, syphilis, leukemia, etc. Usually all these cases are accompanied by more or less deafness and nystagmus.

Hence every case of vertigo should have a complete ear examination, including testing of audition, and usually turning tests, the douche, and sometimes the fistula tests.

In chronic suppuration of the middle ear, vertigo becomes of great diagnostic and prognostic importance, as indicating the necessity of surgery. But one should endeavor to distinguish between irritation of or extension to the labyrinth and extension of the suppuration to the brain.

While local lesions of the labyrinth produce vertigo, it is equally true that any interference in the tracts through which impulses from the labyrinth pass may produce vertigo. Lesions of the pons, the medulla, the cerebellum and its peduncles, and of the eighth nerve itself may involve these pathways. As the auditory nerve passes through the pons and approaches the restiform body it divides, the acoustic bundle passing to the outer side and the vestibular bundle to the inner side of this body. Thus a lesion in this region might involve the vestibular portion, producing vertigo and nystagmus, without involving the acoustic portion and the function of audition. Even lesions of the frontal lobe produce true vertigo sometimes when they involve tracts leading to and from nuclei at the base of the brain, connected with the vestibular neurons. Lesions of the cerebellum through which most of the vestibular impulses pass to the muscular system of the body are most likely to produce incoordination, vertigo, nystagmus and past pointing of a more persistent type than the vertigo of labyrinthine origin.[3]

Instead of direct involvement of these tracts, intracranial disease, by inducing intracranial hypertension, may be the cause of vertigo. Hubby of New York[4] explains it in this way. The saccus endolymphaticus lies within the cranium on the posterior surface of the petrous portion of the temporal bone. Through the ductus endolymphaticus (or aqueductus vestibuli), which passes through the bone to the saccule and utricle, lying in the vestibule, direct connection is established with the fluid circulating in the membranous canals, and pressure within the cranium is transmitted to the labyrinth. If the labyrinth of one ear is more irritable than the other, as it well may be, owing to toxic or local conditions, one-sided labyrinthine impulses are set up and cause the vertigo.

Portman[5] of Bordeaux, France, claims to have relieved cases of this kind, as well as cases of labyrinthine hypertension originating in the labyrinth itself, by opening or destroying the saccus endolymphaticus surgically.

Tumor, abscess, hemorrhage and meningitis are some of the conditions that may interfere with the tracts or cause hypertension. With the vertigo are associated usually, if not constantly, other symptoms which must be interpreted by a competent neurologist. Multiple sclerosis is often associated with vertigo by causing degeneration of the nuclei or tracts which convey vestibular impulses.

In addition to cases of vertigo occurring in demonstrable lesions affecting directly the vestibular apparatus or the neurons distributing its impulses, there is a large number of other cases in which no local lesion can be identified. These are the so-called cases of functional vertigo. They may be mild or severe, temporary or prolonged. Lesions remote from the ear and many systemic conditions are able to affect the labyrinth and cause vertigo. The mode of action may be through the reflexes or due to toxins or circulatory disturbances.

If of a reflex type, as in abdominal disease, it is through the sympathetic nervous system affecting the vasomotor supply of one labyrinth or its connections on one side more than the other. Similar one-sided reflex effects are seen in one-sided pallor, in perspiration of one side of the body and in blanching of one cerebral occipital lobe in transient hemianopsia, etc. Vertigo in diseases of the upper respiratory tract is usually due to a direct extension of the disease to the middle ear involving the labyrinth secondarily. But in a reflex way through the vasomotor nerves disease of the nasal accessory sinuses can produce vertigo without producing demonstrable middle ear disease, illustrated by case 2, reported below. Sluder,[6] I think, makes this clear. He relieved a case of persistent rotatory vertigo in subacute ethmoiditis by anesthetizing the sphenopalatine ganglion. Skillern[7] remarks that vertigo is a frequent accompaniment of sinus suppuration. But in my experience and the recorded experience of others, I think it is rare.

Levine[8] asserts that, in his experience, "the majority of cases of vertigo are due to either a circulatory or a metabolic abnormality."

Vertgio in cardiovascular disease is due to insufficiency of blood supply to the brain and labyrinth. Such diseases are

aortic valve lesions, thoracic aneurysm, heart block, auricular fibrillation, etc. Low blood pressure will produce vertigo. It is common in high blood pressure, especially if the pressure is suddenly lowered. The changes in the labyrinth brought about in these conditions are probably due to changes in pressure of the intracranial contents on the saccus endolymphaticus.

Toxemia causes vertigo through irritation by the poison of the nerve endings in the labyrinth and vestibule. Certain drugs and endogenous poisons, such as are retained in the system in cases of renal insufficiency, etc., are in this way responsible Endocrine disorders have vertigo as a symptom in about 25 per cent of the cases, according to Dana W. Drury.[9] These disorders produce changes in blood pressure and in intracranial and labyrinthine tension. They influence metabolic processes so that a toxin or too much or too little of some normal blood constituent reaches the labyrinth and causes irritation and vertigo. The nerve elements within the labyrinth, like the retina of the eye, are very delicate and easily disturbed by the slightest of influences.

It may be said that these general conditions are outside the domain of otology, but as all cases of vertigo, excepting ocular vertigo, are now held to have their immediate cause in disturbance of the semicircular canals or their connecting neurons, the otologist is called upon more and more to aid in tracing out their etiology. He can do this with more satisfaction to himself, the patient and the physician who asks his aid if he is familiar with all possible etiologic factors and their modus operandi. It may be that many of us have regarded this symptom too lightly in the past, especially in its most obscure phases. If so, it is hoped this superficial review may stimulate more general research in this important field. The most difficult cases to deal with in practice are those in which no fault can be found in any part of the ear.

I would like to report briefly two such cases, in neither of which was there any ear disease.

Case 1. Probably Cerebellar Disturbance.—Mrs. F., aged 49 years, seen August 27, 1927, in consultation with Dr. A. F. Hoge. For two or three months had been having slight dizzi-

ness and unpleasant sensations about the right ear and temple. About ten days before she had a severe attack of vertigo in which everything went round. This continued with remissions and exacerbations, she spending much of her time in bed until the date of the consultation. She stated that all summer and now she felt that she was in perfect health except for the vertigo. Dr. Hoge had given her a very careful and complete physical and general examination. Heart, lungs, abdominal and pelvic organs normal. Blood and urine normal. Metabolism normal. Knee reflexes and Babinski exaggerated on both sides. No Romberg. She walked with a stiff legged, staggering gait, the feet rather wide apart. Mentality unaffected. No headaches. None of the extrinsic eye muscles were at fault. The pupils were normal in size and reacted normally and equally. Fields and acuity of vision were normal. Closing of the eyes had no effect on the vertigo. No nystagmus. Media and fundi of both eyes were normal. Ears showed no abnormality. Both ears normal to hearing and tuning fork tests. Labyrinthine function normal both sides. There was paresthesia of the right side of the face and hyperesthesia of the scalp over the right parietal. The vertigo could not be attributed to the ear. A lesion of the cerebellum was suspected, or of the medulla or pons involving the roots of the vestibular and fifth nerves. As she did not improve, her physician sent her to Dr. Ernest Sachs of St. Louis, who finally performed decompression and exploration of the cerebellum through the occipital bone but found no tumor.

She made a good recovery and has had no vertigo or other unpleasant symptoms since the decompression, which was done some fourteen months ago. She is active in social and household affairs as formerly.

Case 2. Reflex from Cyst in Maxillary Sinus.—Dr. M. E. F., male, aged 42, was attacked with vertigo on September 30, 1928, while playing golf. He played a few more holes but had to be helped home and to bed. The vertigo continued, gradually getting worse. It was of the rotary type. Sometimes he would be nearly normal for a few hours, but most of the time the vertigo was present, whether in bed or out, with eyes closed or open. There was no fever. All body functions normal. No Romberg. All reflexes normal. No headache. No

nystagmus. The Cooper Clinic reported all physical and laboratory findings negative. Blood pressure slightly below normal. Wassermann negative. Eye examination showed normal vision and normal function of the eye muscles. Fundi of both eyes normal. Ears showed normal canals and drum membrane. Normal hearing and normal response to the tuning fork. Turning tests could not be made. Throat normal. Tonsils had been cleanly removed. Nose normal. Accessory sinuses by transillumination unusually bright.

Although there were no focal or paretic symptoms the patient, fearing intracranial trouble, went to Boston and entered Peter Brent Brigham Hospital, at the end of the third week of his illness, where a diagnosis of functional vertigo was made. Intracranial trouble was eliminated.

Not improving, he entered Jefferson Hospital in Philadelphia November 11th. Here he was examined by Drs. M. A. Burns, S. McEwen Smith and F. O. Lewis, whose report I have had the privilege of reading. The X-ray indicated a probable cyst in the floor of the right antrum of Highmore, which inspection and transillumination had failed to indicate. After an exhaustive examination, finding no evidence of other conditions to account for the vertigo, it was decided to open the antrum with a hope but not a promise of benefit to the vertigo. This was done on November 20th, by Dr. Lewis, and a large cyst containing clear sterile fluid removed. From that day to the present, two months, there has been no vertigo, the patient remaining well and strong in every way.

As evidence that the cyst was the reflex cause of the vertigo, it should be recorded that on the second day after the operation the antrum became plugged with mucus and blood clot, accompanied by a severe return of the vertigo. After clearing the antrum the vertigo disappeared completely and at once, not to return. The vertigo was not toxic, for the reason that there was no infection.

Of course, in both these cases it will take time to tell the final outcome. The object of reporting them is chiefly to illustrate the immense difficulties and responsibilities confronting the physician who may be called upon to deal with vertigo.

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XIV.

SOME VERY ACCURATE MEASUREMENTS OF THE AMPLITUDE OF VIBRATION OF TUNING FORK PRONGS.*

By Robert Sonnenschein, M. D.,

Chicago.

I have always been in favor of accurate methods in all functional testing of hearing. In order to have this, it is necessary that instruments which one uses, such as tuning forks, etc., be properly calibrated and that a definite technic be employed in order to get uniform and comparable findings. In the work done with the Webster phonometer, some years ago, it was found that apparently the greatest intensity of vibration emanated from the broad or outer surface of the prongs, a little less from the narrower surfaces, still less from the ends, and least of all from the edges or angles formed by the broad and narrow surfaces. Some forks have prongs which show a quadrilateral cross section so that one cannot say a broad or outer surface if the outer surface and the anterior surface are of the same width. At the time that the tests were made we did not realize that the shape of the prongs (that is, whether quadrilateral or oblong), the distance between the prongs or its relation to the width of the prongs made any particular difference regarding the relative intensity of one or the other side of the prongs. It was not until Mr. Eisenhour of the Riverbank Laboratory called my attention to the possibility that these factors might have some influence that I decided to check the results I had obtained in 1924, in order to definitely settle the question, and also to modify my statement, if necessary, in order that the full truth might be known.

In order to test these points, Mr. Eisenhour and Mr. Tyzzer very kindly instituted some experiments at the Riverbank

*Read as chairman's address, meeting of Middle Section American Laryngological, Rhinological and Otological Society, at Chicago, January 9, 1929.

Laboratory in which I was privileged to participate. In order to definitely measure the amplitude of vibration, the latter was observed through a microscopic eyepiece in which there was a scale upon which the amplitude or excursion of the prongs could definitely be seen. The forks were activated electrically, and the intensity of sound was measured with the sound meter of Mr. Eisenhour. Thus an absolutely accurate picture of the intensity of the sound was obtained and the amplitude of the vibration of the prongs measured. We then used the two very excellent forks that I had employed in my tests with the phonometer, and compared the results obtained with them, with those belonging to the laboratory. In this manner it was seen that the forks which I had employed showed slightly more intensity from the outer surface of the prong than from the narrow surfaces, thus confirming my original findings; but with the Riverbank fork whose shape and the distance between whose prongs were somewhat different from my forks, the reverse of this action was found. In other words, the shape of a fork and the distance between the prongs have considerable influence in determining which surface of the prongs emits the greater intensity of sound. The longer the distance between the prongs, the more sound can escape. In view of the fact that there is only a slight difference in the sound from the outer surface of the prong and that obtained from the narrow surfaces held parallel with the ear, I wish to repeat what I stated in my original paper, that even though in most forks that I have used the outer surface gives slightly more intensity than the narrower one, from a practical standpoint it will make essentially no difference which of these surfaces is presented to the ear, provided that the examiner always uses the same method in all his cases so that he obtains uniform and comparable results.

N. B.—With the phonometer inaccurately tuned, the outer surface of the prongs gives greater force; but with the hearing tube, etc., the two sides of the prongs seem to give the greater pressure. The tables of figures arrived at in the tests are not included but only the conclusions derived from them.

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XV.

ANATOMY, PHYSIOLOGY AND PATHOLOGY OF THE LACRIMAL APPARATUS, MORE ESPECIALLY THE LACRIMAL SAC AND DUCT.*

By Harry Vanderbilt Würdemann, M. D.,

Seattle.

The lacrimal gland secretes a salty, watery fluid which flows diagonally over the eye, keeping it moist, and with the secretion from the meibomian glands lubricates the conjunctival sac. The tears normally pass first through the canaliculi, then into the lacrimal sac, then through the lacrimal canal, emerging by a minute opening into the inferior meatus of the nose, immediately under the anterior third of the inferior turbinated body.

There are few diseases or anomalies of the secretory organ. These are practically limited to adenitis and tumors. Anomalies of the lacrimal passage, however, are common, but these diseases of the apparatus which are not associated with conjunctival, nasal or sinus disease are practically nonexistent. Continuation of a conjunctival affection to the lacrimal passage causing obstruction of the puncta of the canaliculi is constantly noted by the ophthalmologist and readily relieved. It is not likely that infections of the sac causing dacryocystitis are caused from conjunctival disease as they would be very commonly noted in ordinary conjunctivitides, such as blenorrheas and trachoma. It may be said that practically all infections of the lacrimal sac arise from associated conditions in the nasal passages and sinuses, in which the rhinologist is interested and ofttimes effects a cure by nasal treatment or operations.

The lacrimal gland is situated in the upper and outer portion of the orbit in front of the palpebral ligaments, and discharges its secretion upon the conjunctival surface through several ori-

*Read as part of a Symposium on Lacrimal Diseases at a meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., Victoria, B. C., January 16, 1929.

fices behind the retrotarsal fold. It has two parts, the inferior portion at times being so much separated from the superior as to be practically a second gland.

The skull shows a depression behind the nasal process of the superior maxilla, bounded by the anterior ridge, the crysta lacrimale anteriorly, and posteriorally by the crysta lacrimale posterior, which is more distinct, and part of the lacrimal bone, forming the fossa for the lacrimal sac. These two crests unite below to form a narrow inverted arch, which makes the lower part of the lacrimal fossa and its transition into the bony naso-lacrimal canal. The floor of lacrimal fossa, formed half by the lacrimal bone and half by the frontal process of the superior maxilla at its upper and the fossa, is shallower. The bone in this region is very thin.

The lacrimal fossa is directly continuous with the osseous canal, which is formed by the superior maxilla, lacrimal and inferior turbinated bones, the superior maxilla forming the larger part of it. From the posterior part of the frontal process and a part of the body, this makes a gutter which is converted into a canal by the lacrimal bone above and the lacrimal process of the inferior turbinate below, the opening into the nasal orifice or ostium lacrymale being immediately below the line of attachment of the inferior turbinal bone. This canal measures from 3 to 4 mm. in diameter and varies from 12 to 24 mm. in length. In various skulls there will be found variations in the three facial bones which make it. The canal is irregular, being larger above, constricted toward the middle and enlarged below. It may be round or irregularly oval, depending upon the conformation of the skull. In flat-faced people like the Asiatics it is compressed anteriorly; in those with aquiline noses and narrow pupillary distances it is compressed laterally. The direction depends upon the conformation of the facial skeleton. It is influenced by the width of the nasal bridge, the pyriform aperture and the inferior meatus. The usual course is a slight divergence of the canals as they pass down and somewhat backwards from the angle of the orbit mesially between the second bicuspid and the first molar teeth.

The nasal duct is lined with periosteum and mucous membrane; the latter continues with the conjunctiva through the puncta, canaliculi, lacrimal sac and nasal duct and at the ostium

with the mucous membrane of the nose. At the lower portion of the nasal duct there is a flap of mucous membrane forming an imperfect valve, the valve of Hasner. In the canaliculi the lining membrane is scaly, but in the lacrimal sac and nasal duct the epithelium is ciliated, as in the nose. At the junction of the tear sac with the nasal duct a valve has been described by Krause and Biraud. At this place there is either a simple elevation of the mucous membrane or a valvelike structure which may even have a central opening. It must be remembered that the nasal duct is not usually a straight passage, as is illustrated in most anatomic works, but is decidedly curved or may have a bend, usually about the middle portion. The canal may have invaginations of the mucous membrane or even diverticula. This is important to remember in the use of lacrimal sounds, which may readily pass into these invaginations and produce a false passage, with the ultimate danger of atresia in healing or of infection.

The lacrimal puncta are usually single, one above and one below. Multiple puncta have been described. These puncta are not only the openings of siphons but they are opened and closed by the winking of the lids, the muscular pressure of which, as well as the contraction of the tensor tarsi muscle, causes a pumplike suction which forces the fluid from the canaliculi into the sac and downwards into the nose. The passage of this fluid is also aided by capillary attraction and by the bellows-like effect of inspiration and expiration. Radical dilatation, and especially slitting of the canaliculi, immediately negatives most of these functions, leaving only gravitation, capillary attraction and the effect of the air currents in the nose to pull the fluids down. Therefore procedures directed toward securing lacrimal drainage should consider all these mechanical principles.

Obstruction of the lacrimal passages is the first thing that causes disease of the lacrimal sac, aside from systemic disease, such as syphilis or tuberculosis, which are uncommon. The puncta may be occluded by caseous masses from the secretions of the meibomian glands or by foreign bodies, likewise occlusion of the canaliculi and the ostium occurs in congestion of the nasal membrane. The former needs ophthalmic treatment, the latter nasal. Simple dilation of the puncta and canal and

syringing of the sac will remove practically all cases of stoppage of the passages without the necessity of probing. When the latter is resorted to, as large probes as can be passed should be inserted and allowed to remain a considerable length of time in order to open the passage, thereby starting the current of tears to the nose, which, if established for a short time, will secure permanency of the passages. Bony occlusion of the canal is rare; stoppage is nearly always due to mucous membrane hypertrophy and imperfect drainage. If the canaliculi become stopped by detritus, which contains pathogenic pus-producing organisms, then the lacrimal sac becomes affected, first with swelling which may go on to pus production; then the condition of dacryocystitis is produced. This then passes on to ulceration and perforation of the mucous membrane with involvement of the underlying and overlying structures, eventuating in so-called abscess of the lacrimal sac. A mucocele may develop, the contents of which may be expressed by pressure through the canaliculi and puncta, but not usually through the nose, as the obstruction is greater below. If drainage cannot be secured through the nasal duct to the nose, the cure of this condition, from the ophthalmologic standpoint, is an excision of the lacrimal sac by one of the several methods later to be given. Within recent years attempts to effect radical cure of diseases of the lacrimal efferent organs have become much more numerous, but, as Hirschberg expressed it, "In the first third of the nineteenth century Ophthalmology got rid of the idea of making artificial openings into the nasal cavity, which is, indeed, a snare and a delusion." Now, in the first quarter of the twentieth century, the consensus of opinion of the eye surgeons is in favor of extirpation of the sac.

The first cut is through the skin and superficial fascia, when the sac is exposed, the upper portion being covered by the ligament of the tensor tarsi, the palpebral portion of the orbicularis and the dense white subjacent and deep fascia. The anterior lacrimal crest is exposed and after the deep fascia has been removed the delicate posterior lacrimal crest is seen, the free extremity of the sac is firmly held down by the ligament and attached to the periosteum. The lower part is free.

The lacrimal apparatus may be, but seldom is affected by accessory sinus disease. The relations of the sac and the duct

to these sinuses, however, are important when it comes to the radical cure of dacryocystitis by operation. In newborn infants the nasal duct is found 1½ to 2 mm. in front of the anterior end of the antrum. In children of eight months it is one or more millimeters farther forward. The anterior ethmoid cells may be sufficiently forward to form the posterior portion of the bony sulcus of the lacrimal sac. Authors generally contend that the wall of the lacrimal fossa is seldom pneumatic and that the lacrimal sac can never be surrounded by nasal accessory cavities. The morphology shows that the anterior ethmoid cells belonging to the uncinate process and agger nasi occasionally extend into the region of the lacrimal sac and nasal duct. If the frontal sinus is very much enlarged it may extend down as far as the region of the lacrimal bone and enter into relationship with the lacrimal sac. Closure of the nasal duct may be brought about by periostitis of the bone, by foreign body in the nose, dental cysts, syphilitic or tuberculous lesions, by rhinoscleroma, synechiæ between the turbinate and the floor of the nose through periostitis in the neighborhood of the ostium and the formation of scar tissue. Changes in the nose which take up space or cause excessive pressure, such as polypi, bone cysts, cystic swelling of the middle turbinal and spurs and deviation of the septum may close the passage. In atrophic rhinitis extension with atrophy of the bony wall has been observed. Invasion of the efferent lacrimal organs by chronic suppuration of the frontal sinuses, ethmoid cells and maxillary antrum has been described. Some authors have claimed that both phlegmonous inflammation of the lacrimal sac and fistulous processes are preceded by disease of the maxillary antrum, but it is probable that this seldom occurs. What happens is that in antrum suppuration there are associated changes in the nasal mucosa which spread through the nasal duct.

To sum up, it seems to be the consensus of opinion that direct propagation of nasal disease into the lacrimal passages is uncommon, but in the majority of cases diseases of the lacrimal sac are nasal in origin through continuity of the mucous membrane of the nose and that of the tear passages. It may also happen that ethmoid and antrum suppuration may be secondary to suppuration in the lacrimal sac. From the foregoing anatomic, physiologic and pathologic observations, it can

be seen that nasal treatment is indicated in addition to conservative ophthalmologic measures, and that radical surgical procedures should be reserved for cases in which drainage cannot be obtained by such, and that where drainage cannot be secured radical operation should be done, which in the majority of cases means, from the standpoint of the ophthalmologist, a complete excision of the diseased lacrimal sac, if possible, with drainage through to the nose.

The balance of the lecture consisted of the exposition and description of twenty-eight lantern slides made from macroscopic and microscopic specimens by the author, and reproductions of illustrations from Schaeffer, Ramsey, Onodi and specimens from the Army Medical Museum.

XVI.

RETROPHARYNGEAL ABSCESS.*

By Roy A. Barlow, M. D.,

Madison, Wis.

The occurrence of retropharyngeal abscess is sufficiently frequent to warrant a brief discussion of the condition and a consideration of its characteristics. Babbitt aptly states that the associated pathologic condition offers little complexity, but the failure to recognize its existence is often followed by serious and even fatal consequences.

The condition is seen most frequently during the lymphatic or strumous age—that is, early childhood. Wood describes the retropharyngeal collection of lymphatic glands as consisting of the two lymphatic glands back of, or under, the pharyngeal mucosa, in front of the aponeurosis of the tendon and vertebral sheaths, and laterally in close relationship to the great vessels of the neck. These glands connect directly with the deep cervical chain.

Various writers have stressed the etiologic importance of otitis media, accessory sinusitis, tonsillitis and the exanthems. These various centers of infection may cause the suppurative degeneration of the retropharyngeal nodes, but it may be stated broadly that retropharyngeal abscess is most likely due to extension or invasion from upper respiratory infection. It has not been my experience that infection in any one sinus or group of sinuses is more likely to be followed by retropharyngeal abscess than any other. In fact, that the retropharyngeal nodes are involved at all is not so remarkable as that they are not attacked more frequently.

The clinical picture of retropharyngeal abscess is apparently seen most frequently in children of from one to three years

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of age. I have, in the past two years, seen the condition in two patients of about twelve years, one of twenty-nine, and one, a woman, of fifty-seven; all other patients were about two or two and one-half years of age.

The symptoms are those of pharyngeal or postnasal obstruction. There is difficult breathing, even to labored dyspnea: difficulty in swallowing is common, and pain in the ear is frequently noted. Because of encroachment on the resonating column of air in the pharynx the voice or cry simulates a peculiar squawk or brassy sound. Pain in the throat is not constant. The condition of the patient is quite toxic. He usually refuses to lie down, preferring to lie forward or to remain in an upright position. This is no doubt due to the fact that when he lies down on his back the palate falls in contact with the intumescent pharynx and renders breathing more difficult.

Objectively the patient looks ill. He breathes with mouth open. A large swelling is apparent in one side of the neck. The patient chokes easily during examination of the throat, and breathing is almost throttled as the tongue is depressed. This procedure causes a backward and downward pressure which closes the small aperture left by the swollen pharyngeal mucosa. The pharyngeal wall is swollen and bulging forward on one side just behind the anterior pillar. Palpation is most important; if abscess is present there is severe pain to the patient when the examining finger encounters the soft, fluctuating mass.

Treatment for retropharyngeal abscess is incision, and this surgical interference is done preferably in the hospital. The patient is wrapped in a sheet and his head is held by the nurse. The suction apparatus should be in readiness so that when the abscess is incised longitudinally the pus may be evacuated with the suction. If no suction apparatus is available the patient should be placed on his side and, as the incision is made, immediately turned over face down so that the pus may drain. It is well to explore the region with the finger. The massage is instrumental in evacuating residual pus, and in addition the exploration gives the surgeon an idea of the condition of the pharynx.

The after-care is simply that of watchfulness to prevent refilling of the cavity. As a rule, convalescence is short and satisfactory. The cervical adenitis recedes quite rapidly.

When retropharyngeal abscess is not recognized the tumefaction may progress until spontaneous rupture occurs. Frank has called attention to the possibility of mediastinal involvement or burrowing of pus under the fascial sheaths into the mediastinum. Wishart reports a case of erosion of the internal carotid artery with fatal hemorrhage.

The examiner must bear in mind the similarity of the clinical picture of retropharyngeal abscess to aneurysm of the carotid artery, lymphosarcoma, fibroma and cervical Pott's disease associated with erosion of the upper cervical vertebra.

Aneurysm of the carotid artery is not associated with adenitis; there is no fever or leucocytosis. The history is one of gradual development, and on palpation a distinct pulsation may be felt synchronous with the heart beat.

In lymphosarcoma and fibroma the signs and symptoms of acute toxic manifestations are also absent, and on palpation the examiner fails to note the soft fluctuation characteristic of abscess. The cervical glands, if present, are of rock hardness.

Cervical Pott's disease is of longer duration, and roentgenograms reveal destruction of the vertebra.

There is one other condition which, while not identical, should be mentioned, and that is rotary dislocation of the cervical vertebra. In this condition there is a history of trauma; there is considerable pain on movement of the head, and the patient has difficulty in opening the jaws. Roentgenograms show the subluxation of the vertebra and the palpating finger meets with bony resistance.

SUMMARY.

Retropharyngeal abscess occurs frequently enough to warrant our attention. In a small child unilateral cervical swelling in the region of the sternocleidomastoid muscle associated with difficult breathing and swallowing, following an acute respiratory infection, should make us suspicious of the possibility of the presence of a retropharyngeal abscess. Surgical treatment is indicated for this condition.

CONCLUSIONS.

1. Retropharyngeal abscess follows respiratory infection in young children.

2. Unilateral cervical swelling following acute respiratory infection should indicate the possibility of retropharyngeal abscess.

3. Palpation is important in the diagnostic procedure.

4. Treatment is surgical.

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XVII.

COMPLICATIONS OF OTITIS MEDIA AND MASTOIDITIS.*

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In a review of much of the voluminous literature on otitis media and mastoiditis, the writer found many excellent articles describing in detail some of the more important complications, but nowhere could he find a collection of all these complications. In most of the contributions great stress has been laid on the symptomatology, diagnosis and treatment of lateral sinus thrombosis and brain abscess, but little or no mention has been given some of the less common conditions that complicate middle ear disease. The writer has attempted to make such a collection of complications, giving attention only to some of the pathologic features, with some statistical data, and giving practically no mention to the symptomatology or treatment of these complications.

In purulent infections of the middle ear there is an involvement of the mastoid process in a very high percentage of cases. When pus has collected in the middle ear sufficient to cause only a slight bulging, it can be assumed that the pus has already invaded the antrum, and any of the adjacent mastoid cells that communicate freely with it. The middle ear does not have to be full of pus and under tension to cause extension to the mastoid. The antrum is not closed off from the middle ear by any barrier that requires a force or pressure to break down, but is directly connected with the attic of the middle ear through the aditus ad antra, a channel that is always open. If a patient with a purulent otitis media were always in an upright position it might be true that pus would not reach the aditus ad antra until the middle ear was full of pus, as this opening into the antrum is at a high level. However, a patient

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with otitis media does not keep his head erect all the time, and so pus or infective material in the tympanic cavity can easily flow into the antrum when only a small amount may be present.

When infected matter reaches the antrum and adjacent mastoid cells, whether or not an active mastoiditis is set up depends upon a number of factors, chief of which are the virulence of the infective organism, the resistance of the patient and the drainage from the middle ear either through the eustachian tube or through an incision or spontaneous perforation in the drum.

The great majority of such infections of the antrum and adjacent cells recover without an active infection of the mastoid, but others go on to involvement of the cells with the formation of pus and the breaking down of the cell walls. Even this active purulent mastoiditis is not of itself a serious menace to life, but it is the complications that follow or accompany it or the acute otitis media that cause conditions that often result in impairment of the functions of the ear or seriously menace the life of the patient.

Potts[1] found in a series of 49 consecutive cases of mastoiditis following middle ear infection that 48.9 per cent developed some type of complication. Of this number 16.3 per cent were serious intracranial complications with a mortality of 6.1 per cent. It has been found that most intracranial complications are much more frequent when the mastoid infection is due to a streptococcus and are much more likely to follow a chronic than an acute infection.

Ballenger[2] mentions that Hegener studied 5,000 cases in the Munich clinic and found that in one-half of 1 per cent of infections of the middle ear intracranial complications resulted.

In a study of the intracranial complications of otitis media observed at the Cook County Hospital during eleven years Yerger[3] found that 10 per cent of the total number of cases of suppurative otitis media had some form of intracranial involvement.

Swift[4] states that in a series of 800 cases of mastoiditis operated upon in the Leed's Infirmary during a year and a half, and reported by Munby and Jewett in the British Medical Journal for July, 1926, the incidence of complications was 147, or 18.1 per cent. He also states that in 1926, in six of the hos-

pitals of Seattle, 123 mastoid operations were done, with 12 complications. In the Leed's series brain abscess occurred in 8.9 per cent of all cases; lateral sinus thrombosis in 5 per cent; purulent meningitis in 2.5 per cent; while in the Seattle hospitals 10 per cent were lateral sinus thrombosis and the others less than 1 per cent each.

Neumann[5] found that 88 per cent and O'Koda 81 per cent of the cases of intracranial involvement were preceded by a chronic suppurative otitis media.

In a statistical study of 56,324 patients with various diseases of the ear Rugg[11] found 86 deaths due to otogenous complications. Therefore 0.15 per cent of all ear ailments proved fatal. He also found that out of 28,378 cases of acute and chronic otitis media there were 86 fatalities due to otogenous intracranial complications, or a percentage of 0.3.

In considering the spread of infection from the tympanic cavity there are many factors to be considered. According to Beck, as quoted by H. Friedenwald and M. L. Breitstein,[6] these factors may be grouped as follows:

1. Loci minores resistance which occur normally.
2. Unusual clefts and fissures which occur anatomically or which have not closed.
3. A breaking down process of the surrounding bone.
4. The lymph drainage of the middle ear.

Under the first heading there should be considered the fibrous tissue connection between the bony and cartilaginous canal, the incisuræ Santorini in the cartilaginous canal, the thinness of the posterior superior wall of the bony canal, the vascular communication between the auditory canal and the mastoid cells, the close proximity of the parotid gland, the variation in thickness of different parts of the mastoid cortex and the degree of pneumatization of the mastoid process.

This development of the mastoid cells has much to do with the complications following their infection. It has been found that there is a great variability in the amount of pneumatization of the mastoid in different people. While ordinarily the mastoid process is undeveloped and has not become pneumatized in infants, it has not infrequently been found that a child under a year old has quite extensive development of the cells.

On the other hand, in some adults only a part of the process is ever invaded by cells. In such cases there might be no cells close enough to the sigmoid sinus to cause its infection by direct extension. In other cases with an extensively pneumatized mastoid, the cells may be in close proximity to the sigmoid sinus, the dural covering of the brain, the facial canal, the internal ear structures, and other cranial nerves, so that an infection would be more likely to cause complications in these structures.

The variation in the thickness of different parts of the mastoid cortex has much to do with the extension of an infection into the tissues of the neck to form cervical abscesses. This will be taken up more in detail later on.

In considering the unusual clefts and fissures, there is found most commonly a patency of the squamous-mastoid suture. There may also occur dehiscences in the wall between the auditory canal and the antrum. More rarely there are dehiscences in the floor of the antrum, the tegmen tympani and in the posterior wall of the carotid canal. These defects are of importance in the transmission of infection to the important structures adjacent to them.

The factor of the breaking down process of the surrounding bone is to be considered chiefly in a study of the type of infecting organisms. Those of greater virulency cause a quicker and more complete breaking down of the bony tissue and therefore are more apt to cause complications. The most virulent type of organisms has been found to be the streptococcus mucosus capsulatus, while next in order comes the streptococcus hemolyticus.

In considering the lymph drainage of the middle ear, there are two possible ways for lymph drainage to occur in otitis media, according to Most:[7]

1. Through the drum to the lymphatics of the external auditory canal and pinna;

2. Through the lymph vessels of the eustachian tubes to the glands in the lateral retropharyngeal walls.

Complications following mastoid infections depend to some extent also on the type of mastoiditis. Kopetsky[8] has pointed out that the infection of the mastoid may occur as the coalescent or the hemorrhagic type. The former occurs with the

formation of pus within the cellular structure and the breaking down of the cell walls to form an abscess cavity within the mastoid cortex. This type of mastoiditis is much more apt to give rise to perisinus abscess, lateral sinus thrombosis, extradural abscess and cervical abscesses than are cases of hemorrhagic mastoiditis.

This latter type consists of an involvement of the mucous membrane lining the mastoid cells, which becomes thickened and swollen with intense congestion of the vessels and the formation of small thrombi within the venules. There is no breaking down of the cell walls, and the cell spaces contain no pus but are usually filled with blood-tinged serum or blood. The formation of the thrombi is Nature's attempt to localize the infection to the mastoid cells. At first the thrombi are of the noninfective type, but they may easily become infected because of the close contact with the causative microorganism present within the mastoid cells, and may then give rise to systemic infection or may break down to form pus. This type of mastoiditis gives rise to systemic infection earlier than the coalescent type and without lateral sinus thrombosis, but is less apt to result in extradural and cervical abscesses.

LATERAL SINUS THROMBOSIS.

Statistics on the involvement of the lateral sinus following mastoiditis vary greatly. Some observers claim it is much more common in those cases due to a chronic otitis media and mastoiditis, while others state it is seen oftener in acute infections. Fraser's[9] statistics, which are in accord with those of the majority of European observers, show that 75 per cent of cases of sinus thrombosis follow chronic infections of the middle ear and mastoid. On the other hand, in the forty cases reported by Atkins,[10] 75 per cent followed acute suppuration of the middle ear, while the remainder occurred in chronic suppurations of which the majority were of a cholesteatomatous nature. His series also showed that 70 per cent of the cases were in males and 85 per cent occurred on the right side. These figures correspond to the general observation that sinus thrombosis is more common in males and on the right side.

However, Rugg,[11] in a study of 422 otogenous brain complications, has found these complications as a whole to be

equally divided on both sides. He also found that in 3,176 cases of otitis, acute and chronic middle ear infections were divided equally between the right and left side.

Statistics on the incidence of sinus thrombosis accompanying mastoiditis are also somewhat at variance. White[12] stated that the fifty-first annual report of the Manhattan Eye, Ear and Throat Hospital gave a series of 566 cases of acute and 22 of chronic mastoiditis, with only 11 cases of sinus thrombosis. He quoted Welty as reporting three cases of sinus thrombosis occurring in a series of 100 cases of acute mastoiditis.

Kopetsky's[8] figures show a much higher incidence of sinus thrombosis. He reported 25 cases of sinus thrombosis, or 20 per cent, in 126 cases of coalescent mastoiditis, and 6 cases, or 30 per cent, in 20 cases of acute coalescent mastoiditis superimposed on chronic purulent otitis media. In his analysis of 44 cases of hemorrhagic mastoiditis he found 15 cases of sinus thrombosis, or 34 per cent.

Most authors, however, agree that lateral sinus thrombosis occurs in from 3 to 6 per cent of the operated cases of mastoiditis. Naftzger[13] reports 6 cases in 192 (3.12 per cent), and quotes Hill as having 7 cases in 166 (4.21 per cent); Downey, 5 cases in 79 (6.32 per cent); Gerber, 25 cases in 524 (4.77 per cent); and the Massachusetts Charitable Eye and Ear Infirmary, 19 cases in 497 (3.82 per cent). Statistics from the Mayo Clinic[14] show that during a period of five years there was found 19 cases in over 500 (3.8 per cent).

Yerger[3] reported a series of 34 cases of sigmoid sinus thrombosis, which was 3 per cent of the cases of suppurative otitis media.

In the development of lateral sinus thrombosis there is usually a history of a middle ear infection, but this infection may have been slight and the ear may have returned to normal within a few days. Some time later there may be a sinus thrombosis either with or without a demonstrable mastoiditis. Many of such cases as these have been diagnosed as primary mastoiditis and primary sinus thrombosis. Jugular bulb thrombosis may occur as a complication of acute middle ear infection, in the absence of any involvement of the mastoid cells, by the transmission of infection from the middle ear through

a dehiscence in the floor of the tympanic cavity directly to the bulb.

Lateral sinus thrombosis is seen more often after a mastoid operation has been performed than at the primary operation. Sometimes it is caused accidentally at the time of the mastoid operation by an injury to or penetration of the vein by an instrument or spicule of bone. In most cases, however, the thrombosis had probably commenced before the mastoid operation was done and only developed into a state to be recognized some time after the operation.

The delayed or incomplete mastoid operation is very frequently the underlying cause of sinus thrombosis. However, thrombosis often follows early and complete mastoidectomy, while many cases of perisinus abscess do not lead to sinus infection and thrombosis. It occurs most often as a result of necrosis of the overlying bony plate, with first involvement of the outer wall of the vein, then extension of the infection into the lumen and finally thrombosis. It frequently occurs also by the extension of a thrombosis to the sigmoid sinus from the small thrombi in the venules of the mucous membrane of the mastoid cells and intercellular bony framework without any necrosis of bone. Such a process as this is sometimes seen in children, in whose mastoid process there is apt to be but little pneumatization and a relatively large amount of diploe. There is an infection of the small emissary veins of the diploe which easily can become completely closed off by thrombus formation and may extend into the larger veins and finally the lateral sinus. A similar process is seen more often in adults with the hemorrhagic type of mastoiditis. In these types of infection it is important to remember that the infection and thrombosis of these small venules and emissary veins within the mastoid structure will give a septicemia with symptoms identical with those of lateral sinus thrombosis when the lateral sinus has not as yet become involved. Such cases require only a simple mastoidectomy and need no exposure of the sigmoid sinus or ligation of the jugular vein.

Hertz[15] contends that sinus thrombosis is often present in infants but is not recognized because of the concomitance of such conditions as pneumonia, meningitis and gastroenteritis,

which he thinks are often embolic in origin, the focus being the infection in the lateral sinus.

There is some variation in the location of the sigmoid sinuses, which, however, seems to have but little influence on the frequency of its involvement in mastoid infections. Sometimes the sinus is well posterior in the mastoid process, with few or no cells immediately adjacent to it. At other times it is far forward, close to the posterior wall of the external auditory canal. In other instances it may be situated deep within the bone and covered by a thick cortical layer and often overlapped by mastoid cells. The opposite may occur where the sinus is covered only by a thin cortex, and there may even be dehiscence in the bony plate overlying the sinus. It has been found that the percentage of sinus infection in cases where the lateral sinus is impinging on the antrum and posterior canal wall is no greater than in those cases having a broad, well-developed mastoid process with the lateral sinus more posteriorly situated. The proximity of the mastoid cells to the sinus and the occurrence of dehiscences, however, do have some influence on the frequency of infection of the sinus. Statistics show that the right lateral sinus is larger than the left, and its relationship to the mastoid cells is such that it is rendered more liable to infection than the left, in the proportion of 3 to 2.[16]

The free communication of the intracranial venous sinuses allows the possibility of a wide extension of a thrombosis beginning in the sigmoid sinus. The right lateral sinus is continuous with the superior longitudinal sinus, while the left is continuous with the straight sinus. There is usually a communication between the right and left lateral sinuses, either through a small communciating branch or through a fenestra in their walls. A thrombus may therefore extend from one sigmoid sinus up to and beyond the torcular herophile into the lateral sinus of the opposite side; it may extend into the superior longitudinal sinus or into the straight sinus. Its downward extension may involve the petrosal and cavernous sinuses, the jugular and facial veins and may extend to the subclavian vein.

Mention might be made of thrombosis of the condyloid veins, which has occurred in some cases. Infection of the anterior

vein may cause extension to the occipitoatlantoid joint, and infection of the posterior vein may give rise to abscesses between the cervical vertebræ and the deep muscles of the neck, causing fixation and contraction of the neck.

Thrombosis of the lateral sinus occurs most often at the knee of the sigmoid portion, and next in frequency at the jugular bulb. It may exist as a mural clot occupying only a small area on the inner wall of the vessel or it may be of the obliterating type that completely fills the lumen of the vein. The thrombus may be infected from the start, giving early the symptoms of a general systemic infection, or the clot may be entirely sterile and its formation be accompanied by no symptoms. Again the clot may be infected, at first giving a bacteremia, and later the patient may show the disappearance of the symptoms of septicemia because of the further progression of the thrombus to the obliteration of the vessel, with the consequent prevention of the septic material getting into the circulation.

At times the sinus wall will be found at operation to have broken down and will be seen discharging pus into the mastoid wound, while the pus is prevented from getting into the circulation by the sterile extremities of the thrombus entirely closing off the vessel. Sometimes cases of lateral sinus thrombosis will recover spontaneously with disappearance of the preliminary middle ear or mastoid infection and organization of the clot.

The infected thrombus may suppurate and disintegrate, throwing showers of virulent organisms and septic emboli into the blood stream. This frequently gives rise to metastatic abscesses in the joints, lungs, kidneys, liver, spleen, brain and surface parts of the body.

At other times the thrombus may rupture into the cranial cavity, causing a cerebellar abscess or a purulent meningitis. Occasionally there is complete organization of the thrombus with later recanalization and restoration of the circulation through the sinus.

SEPTICEMIA.

While septicemia may more properly be regarded as a symptom of sinus phlebitis or thrombosis rather than a com-

plication by itself, it has been thought advisable to treat it separately as though it were a distinct complication.

In the septicemia accompanying middle ear or mastoid disease it is not the presence of the bacteria in the blood that gives an increased menace to the patient's life. The menace is chiefly in the possibility of the formation of metastatic lesions in the lungs, liver, kidneys, brain, etc.

The entrance of bacteria into the circulation is a rather frequent occurrence, being commonly seen in several diseases, such as pneumonia, malaria, typhoid fever and influenza. The presence of these organisms does not in itself complicate the primary disease. The blood is usually perfectly competent to take care of these foreign bodies and rapidly eliminate or kill them. The bacteria do not circulate and multiply in the blood stream, but are in all likelihood there only a few minutes, being rapidly destroyed by the antibacterial agents of the blood and by the reticuloendothelial system of cells located in certain tissues of the body, such as the bone marrow, spleen, liver and lymphatics.

In septicemia of the middle ear, mastoid and lateral sinus infection the bacteria are discharging intermittently and irregularly into the blood stream from the focus of infection. It is the reaction of the body to these invading organisms that gives rise to the characteristic symptoms of lateral sinus thrombosis, such as chills or fever. When the temperature in these cases has again reached normal it is probable that the invading organisms have all been killed. The rapidity of the death of the circulating microorganisms may account for the fact that 40 to 60 per cent of cases of lateral sinus thrombosis with symptoms of systemic infection show a negative blood culture.

Septicemia is probably much more common in acute middle ear conditions than is generally supposed and is more likely to be met with in children than in adults. The symptoms and local signs of ear involvement may be mild in character and may entirely disappear with the onset of the septicemia, so that there is some danger of failure to recognize the middle ear infection as a cause of the systemic illness. According to Kopetsky,[8] in the hemorrhagic type of mastoiditis, systemic involvement occurs as soon as the mastoid becomes infected.

The treatment of otitic septicemias is, of course, first the eradication of the primary source of infection. This means a thorough mastoidectomy and in many cases obliteration of the lateral sinus and ligation or excision of the jugular vein. Secondly is the intravenous treatment, an important part of which is the use of blood transfusion to supply new hemoglobin and bactericidal agents.

METASTATIC ABSCESSES.

In most cases of septicemia the blood and the cells of the reticuloendothelial system are capable of destroying the invading bacteria and preventing their gaining a foothold in any other part of the body. However, if the infection is too much prolonged, or the resistance of the patient too weak, or the virulence of the infection unusually great, there comes a time when the phagocytic cells are partly destroyed and the bactericidal substances in the blood become less active so that bacteria remain longer alive in the blood. It is then that certain of them may gain a foothold and begin to grow in various parts of the body. Secondary centers of infection are thereby formed and from these more bacteria and toxins enter the circulation.

When such abscesses occur they will need to be opened if they are in accessible locations. When they are in the kidneys, heart or other internal organs the best that can be done is careful nursing and medicinal treatment. If recovery takes place the organs that have been affected are apt to be much impaired in their subsequent function.

Metastatic lesions may occur early or late in the course of sinus phlebitis. Metastatic abscesses are quite common, especially in the skin, joints, muscles and synovial membrane. In Atkins'[10] series of 40 cases of sinus thrombosis metastases occurred in 40 per cent, the joints being most commonly involved. Metastases are not dependent on the breaking down of the thrombus and the spread of the infected particles in the general circulation but the bacteria themselves escape into the blood stream. A metastasis may exist in spite of all negative blood cultures. It may act as a "fixation abscess" which tends to clear the blood stream.

The distribution of metastatic lesions depends probably largely on the type of organism causing the infection and its inherent selectivity. The organisms in the blood stream exert a selective action for certain types of tissues. According to Kopetsky,[17] it has been found that the pneumococcus and streptococcus mucosus capsulatus invariably affect the endocranium after invading the mastoid. Streptococcus hemolyticus shows several strains, one selecting the joints and serous membranes; another, the superficial structures, such as the skin and muscles, while still another attacks the lungs. Giesswein,[18] however, states that there is no difference between the metastases in acute and chronic middle ear diseases.

CAVERNOUS SINUS THROMBOSIS.

Bowers[19] states that cavernous sinus thrombosis as a complication of mastoiditis is rare. His search of the literature revealed only 26 cases secondary to mastoid infection. He found that 52 autopsies had been reported in which cavernous sinus thrombosis was associated with an aural condition. This would leave 26 cases which originated from ear conditions other than mastoiditis. In those cases associated with mastoid infection the onset was from 12 hours to 25 days after operation, the average being about two days. The duration of the illness after its recognition was from two days to several weeks, the average being about three days. The organism found in most cases was the streptococcus. In this condition the blood culture is apt to be negative.

The most common route from the ear to the cavernous sinus is through the superior and inferior petrosal sinuses.

Next to disease of the sphenoid sinus pyogenic infection from the ear is the most common cause of thrombosis of the caverous sinus. Dorland Smith,[20] in an analysis of 140 cases, found the original source of infection to be in the ears in 40 per cent, orbit and face 35 per cent, mouth and throat 13 per cent, nose 9 per cent, and other causes 3 per cent.

The prognosis is nearly always fatal. Three or four cases have been reported of recovery following ligation of the jugular vein and drainage of the lateral sinus. One case recovered following drainage of the cavernous sinus posteriorly through

the petrous bone. Among all cases of cavernous sinus thrombosis about 15 have recovered spontaneously.

BRAIN ABSCESS.

Brain abscess is not an infrequent complication of middle ear and mastoid disease. It has been found that about 85 to 90 per cent follow the chronic form of middle ear infection. The most susceptible age is between the tenth and thirtieth years of life, and men are more frequently affected than women.

Ballenger[2] states that Jansen, in the Berlin Ohrenklinik, found one brain abscess in 2,650 cases of acute, and six in 2,500 cases of chronic suppurative otitis media. He says that 91 per cent of those brain abscesses that complicate otitis media are associated with the chronic form and 9 per cent with the acute. He states that Hermann analyzed 570 cases of brain abscess and found 457 due to chronic middle ear infection and 113 due to the acute form.

Frasner,[21] at the Edinburg Royal Infirmary, reports that among 2,000 cases of acute purulent otitis media there was one case of cerebellar abscess and three of temporal lobe abscess. He also reports that among 6,492 cases of chronic middle ear suppuration there were 26 cases of cerebellar abscess and 27 cases of temporal lobe abscess.

In Yerger's[3] study of intracranial complications, brain abscess was found in 2 per cent of all the cases of suppurative otitis media.

Hirzinger,[22] in reviewing cases of brain abscess, found that out of 28 cases, 6 originated from acute otitis and 22 from chronic otitis. Of these 28 cases, 4 were in the cerebellum and 24 in the cerebrum.

The great majority of otitic brain abscesses are located in the temporosphenoidal lobe. The cerebellum is second in frequency of attack, and the pons and crura cerebri are comparatively rare locations for abscesses. Statistics show that out of 1,400 cases approximately 70 per cent involved the cerebrum and 30 per cent the cerebellum. Of 18 cases of otitic brain abscess studied by Yerger,[3] 65 per cent were temporosphenoidal, 23 per cent occipital and 12 per cent cerebellar.

Most brain abscesses are found adjacent to diseased bone and are due to direct extension of the infection from the part

of the ear involved to the brain tissue. The most frequent avenue of entrance is through the tegmen tympani, therefore usually resulting in an abscess located in the temporosphenoidal lobe. Infection of the brain may take place following sinus thrombosis. Blezl[23] pointed out that thrombosis may lead to formation of brain abscess in one of three ways: (1) by continuity, (2) by the metastatic-embolic route; (3) by retrograde transportation of infectious material from the sinus into the meningeal vein. He believes that continuity should be considered, especially in cerebellar abscess; that metastatic embolic abscess is usually situated in the marrow substance and the retrograde type in the cortical substance. An abscess following thrombosis of the superior petrosal sinus will be located deep in the temporal lobe or cerebellum.

Cerebellar abscesses result from extension through the labyrinth or by way of the sigmoid sinus, the majority being through the labyrinth.

When a brain abscess is the result of direct infection there will often be found a track leading from the mastoid wound to the abscess cavity. If it has occurred through the blood stream or lymphatics no track will be found. When occurring through transmission by the blood stream there may be multiple abscesses, although these are quite rare and usually occur in cases of pyemia.

Abscesses may be encapsulated or may not have a retaining wall. The chronic abscess usually has a distinct capsule which varies somewhat in thickness. The walls of these abscesses are primarily granulations which gradually become indurated and change into cicatricial tissue. The walls surrounding a brain abscess sometimes reach a thickness of three-eighths of an inch, the thicker wall usually indicating an abscess of longer duration.

The abscess cavity is filled either with actual pus or with a grumous material made up of fibrin, serum and leucocytes with a few pus cells. The unencapsulated abscesses usually contain a thin, foul-smelling pus mixed with broken-down substance. The unencapsulated abscess may be the result of rupture of the wall of the neighboring encapsulated abscess. In the latter type the brain tissue is only slightly affected, while in the unencapsulated variety there is a destruction of brain

substance of varying degree, often to a large extent, and sometimes the entire hemisphere is softened and swollen, and punctured with hemorrhagic points.

PERISINUS ABSCESS.

The bony plate covering the lateral sinus is of a more firm and solid texture than is the intercellular framework or the diploic structure of the mastoid process. Therefore considerable destruction of the softer bony tissue of the mastoid can take place before the sinus plate becomes eroded.

The erosion of this plate depends largely upon the type of organism causing the mastoid infection. The streptococcus mucosus capsulatus is apt to cause a rapid erosion of bone, and the sinus may rapidly become infected, with thrombus formation. On the other hand, organisms of less virulency may more slowly cause necrosis of the sinus plate and the formation of pus against the sinus wall. This perisinus abscess may be in contact with the sinus sometimes for a considerable time without infection of the inner lining of the vessel or the formation of a thrombus.

Perisinus abscess sometimes seems to have no direct connection with the tympanic antrum. However, the perisinus region, in probably all cases, was infected by a direct continuation of the infection from the antrum through any intervening mastoid cells. The infection in these intervening cells may have subsided with a drainage of the pus back into the middle ear cavity, leaving the contaminated area adjoining the sinus to develop into an abscess. A perisinus abscess can spontaneously evacuate itself by draining into the middle ear, by perforating the mastoid cortex or by spreading along the mastoid emissary vein to reach the surface of the skull.

There have been reported two cases of torcular subperiosteal abscess in which the abscess formed from an infected thrombosis at the torcular.

EXTRADURAL ABSCESS.

The extradural abscess is by far the most frequent otogenous intracranial complication. Rugg[11] found it 251 times, among 422 otogenous intracranial conditions.

This condition usually occurs in an acute exacerbation of a chronic purulent otitis media and mastoiditis. It is caused by

the necrosis of the bony plate covering the dura, most often at the tegmen tympani and tegmen antri. These abscesses are therefore generally located in the middle fossa of the skull.

The communication between the extradural abscess and the diseased middle ear may be very small or it may become entirely occluded.

An infected thrombus of the lateral sinus also may give rise to an extradural abscess, in which case it will be against the dura overlying the cerebellum. It most often occurs as a perisinus abscess and therefore is often associated with sinus phlebitis and thrombosis. This condition is usually unrecognized before operation and complicates little, if any, the mastoid infection.

In rare cases extradural abscesses may form on the posterior or anterior side of the temporal pyramid. Those that develop on the posterior side result from chronic suppuration of the middle ear when the labyrinth is also involved. Those that occur on the anterior surface of the pyramid arise from direct involvement of the veins in the pneumatic cells of the tip of the temporal pyramid, in acute cases of suppurative otitis media.

SUBDURAL OR INTRADURAL ABSCESS.

This type of abscess is characterized by the extension of the infection through the dura, with the formation of the abscess between the dura and the arachnoid. It is usually well walled off from the brain, but is a more serious menace to life than is the extradural abscess, because the arachnoid is a more delicate membrane than the dura. Subdural abscesses may exist for considerable time without producing serious symptoms. The accumulated pus, however, unless released by operative measures, will eventually break into neighboring structures. Usually they empty themselves back into the middle ear spaces, but the infection may also penetrate into the subarachnoid spaces, giving rise to a leptomeningitis.

PERILABYRINTHITIS AND LABYRINTHITIS.

These conditions occur only rarely as complications of purulent otitis media or mastoiditis. Kopetsky,[8] in an analysis of 126 cases of coalescent mastoiditis, found only 0.5 per cent

accompanied by a labyrinthitis. In Yerger's[3] series there were 10 per cent of intracranial complications in the total number of suppurative otitis media cases, and of this number there were less than 1 per cent of cases of manifest labyrinthitis.

A. Logan Turner[24] of Edinburgh has recently made a most detailed study of labyrinthitis. He states that during the years 1907 to 1925 (inclusive) 10,673 patients suffering from middle ear suppuration sought advice of the Ear and Throat Department of the Royal Infirmary at Edinburgh. Of these, 2,660 were diagnosed as cases of acute or subacute inflammation and 8,013 were classified as cases of chronic otorrhea. Of these, a secondary affection of the labyrinth was diagnosed in 150 patients; in 21, the labyrinthitis complicated acute or subacute otitis media, and in 129 the chronic form of suppuration. Labyrinthitis was, therefore, found to occur in 0.7 per cent of the acute, in 1.6 per cent of the chronic and in 1.4 per cent of the total cases.

In this series circumscribed labyrinthitis occurred in 46, or 30 per cent; acute serous labyrinthitis in 3, or 2 per cent; acute purulent labyrinthitis in 24, or 16 per cent; chronic purulent labyrinthitis in 61, or 40 per cent. Spontaneous cure of the labyrinthitis with loss of function occurred in 10 per cent.

Turner quoted Friedrich as stating that a labyrinth complication develops once in every hundred cases of middle ear disease. He also mentions that Whitehead's figures show that out of 691 cases of suppuration, the labyrinth was involved in 27, or 3 per cent; and that Uffenorde found, in a series of 3,135 cases of chronic middle ear suppuration, that the labyrinth was diseased in 105, or 3 per cent.

Occasionally in a middle ear infection, especially when there is an extensive pneumatic development of the mastoid process in the region of the posterior and horizontal semicircular canals, there will be invasion of these cells immediately surrounding the bony labyrinth, giving rise to a perilabyrinthitis. This condition may easily be mistaken for a true labyrinthitis and can only be distinguished from it by the accurate observation and interpretation of the accompanying nystagmus and the reactions to the caloric tests.

True labyrinthitis is characterized by an involvement of the end organs of the cochlear and vestibular nerves. They may

become involved by a necrosis of the promontory on the inner wall of the middle ear, infection through the foramen ovale or rotundum, erosion through the wall of one of the semicircular canals, or the infection may reach the labyrinth and spinal fluid suddenly by the blood stream or lymphatics. In the latter type of cases there will develop a rapidly fatal labyrinthitis and meningitis. In the former types, due to the slower extension of the infection, there will be time for protecting adhesions to form to prevent contamination of the meninges. When labyrinthitis occurs during an acute otitis media the pathway of infection is usually through the oval window. In chronic middle ear disease the entrance is through the external semicircular canal in the great majority of cases.

Infection of the labyrinth through the blood stream is rare. When it does occur it is usually in patients with syphilis. However, it can also occur by metastasis from a middle ear or mastoid infection through the vascular connection between the lateral sinus, the petrosal sinuses and the labyrinthine veins.

Labyrinthis may also occur by invasion from a meningitis of the acute purulent form or from the epidemic cerebrospinal type. Infection of the labyrinth, in these cases, occurs by invasion of the aquæductus cochleæ and then involvement of the lymph channels and acoustic nerve. This occurs mostly in children and is apt to result in deafmutism.

A purulent infection in the labyrinth may affect the whole membranous labyrinth or involve only a part of it. When the horizontal semicircular canal is invaded it is common to find only the contaminated part infected, and the rest of the labyrinth walled off from the infection.

In the majority of cases the purulent process is prevented from reaching the cranium by the formation of adhesive processes in the perineural and perivascular lymph spaces. This cuts off all communication between the labyrinthine fluid and the brain or meninges.

In some cases, especially in the more severe types of infection, there has not been time for these protective barriers to form, so that a diffuse labyrinthitis results, and possibly also a meningitis or cerebellar abscess.

It has been found that the labyrinth may become involved at nearly any time during the course of otitis media. In acute

or subacute infections Turner found the average onset to be at the end of the third or at the beginning of the fourth week of the otitis media. In chronic suppurations the labyrinth may be invaded at any time.

The presence of cholesteatoma in the chronic suppurative cases makes the labyrinth more liable to secondary infection, as it causes a slow erosion of the outer bony capsule of the internal ear. However, erosions and fistulæ may occur in the absence of cholesteatoma by the action of the suppurative process itself.

Circumscribed labyrinthitis is usually localized in the region of the lateral canal. The diffuse or generalized forms of labyrinthitis give rise to the sudden characteristic symptoms. The diffuse acute types are seen more often in the acute otitis medias than in the chronic. When the acute stage has subsided and the condition has passed over into the chronic latent purulent type of labyrinthitis the distressing symptoms are relieved. The latent stage develops more often in those cases that complicate chronic purulent otitis media.

MENINGITIS.

Meningitis is a rather frequent complication of otitis media and mastoiditis. In Yerger's[3] study of complications of suppurative otitis media he found meningitis in 5 per cent, of which the mortality was 97 per cent. Kopetsky[8] found 3 per cent in his 126 cases of acute coalescent mastoiditis, 15 per cent in 20 cases of acute coalescent mastoiditis superimposed on chronic purulent otitis media, and 5 per cent in 44 cases of hemorrhagic mastoiditis.

Clinically there have been described three types or degrees of meningeal irritation (Yerger). The mildest type has been called meningismus. It gives rise to but mild meningitic symptoms and does not change the pressure or character of the spinal fluid. An example of this type is seen in the mild meningitic symptoms occurring at the time of the primary occlusion of the sigmoid sinus or jugular bulb by infection or thrombosis.

The second type is serous meningitis, sometimes spoken of as the circumscribed, sympathetic or protective type. It occurs when a suppuration adjacent to the meninges has set up an

active inflammation of the meninges of moderate severity. It may be due to the pathogenic organisms themselves or to their toxins. The involved area of dura may be only hyperemic or it may be deeply congested and covered with granulations. The inflammation may spread through the dura to its inner visceral side, where it may become circumscribed or go on into a lepto-meningitis. In this type there are more pronounced meningitic symptoms, and there will be a change in the spinal fluid. The pressure will be raised and the cell count will be increased up to 10,000 per cm. The spinal fluid, however, will remain sterile and the symptoms will disappear if there is an early drainage of the suppuration adjacent to the meninges.

In cases that have not had early or sufficient drainage there will be found borderline cases that are passing over into the third or purulent or diffuse type of meningitis. When this stage is reached the spinal fluid pressure is still higher, the cell count becomes greater (over 10,000), the culture of the fluid is apt to be positive, and the meningitic symptoms become pronounced and unmistakable. While cases of the first two types recover, as a rule, those of the third type are practically always fatal.

Serous meningitis accompanying ear infections may be due to extensions of the infection through the tegmen tympani and antri or through the labyrinth. It more often follows chronic otitis media and mastoiditis than the acute form. This condition may go on to purulent meningitis or may subside without the cerebrospinal fluid becoming infected. Many cases are prevented from progressing by the early operative treatment of the middle ear or mastoid suppuration.

Diffuse purulent meningitis of otitic origin may be due to perforation through the tegmen tympani or antri, or the carotid canal, by extension from the labyrinth by way of the internal auditory meatus, through the aquæductus vestibuli or the aquæductus cochlea, as the terminal stage of a brain abscess, and through extension by way of the lymph or blood stream. In the lattter method of invasion many observers believe the infection reaches the subarachnoid area by traversing the lymph spaces which surround the nerves and arteries. In infants and young children infection may also reach the meninges through one of the open sutures of the temporal bone.

Extension by way of the tegmen tympani may occur more readily in young children because of the thinness of this dural plate. In mastoid disease the organisms may reach the meninges through the small diploic veins or the lymphatics. In cases of brain abscess meningitis may occur from extension through the brain tissue to the ventricles before a distinct capsule has formed around the abscess.

The termination of the purulent type of meningitis is nearly always death. Dench[24] reports some favorable results from the exposure of very large areas of dura in the middle cranial and cerebellar fossæ after the performance of a simple mastoid operation in acute cases, or by a radical operation in chronic cases. There is one type of meningitis that runs a longer course than the common purulent form. It usually complicates chronic middle ear suppuration with involvement of the labyrinth. There may be a number of remissions in the symptoms, sometimes of several months, but finally an attack terminates in death.

FACIAL PARALYSIS.

Involvement of the facial nerve in acute middle ear or mastoid infection can occur from extension of the inflammation along the course of the facial nerve, whether in the facial canal, in the internal auditory meatus, or as a result of bone destruction within the mastoid itself. In acute mastoiditis paralysis may be due to an edema of the nerve caused by absorption of the toxins from the adjacent infective process. This type of case is promptly relieved by a mastoidectomy.

In cases of chronic mastoiditis there may be necrosis of much of the bone close to the facial nerve without causing disturbance in the function of the nerve. The degree of facial paralysis depends upon the severity of the inflammation and whether the entire nerve or only certain bundles in the nerve are affected.

One sometimes finds the paralysis more pronounced in the upper branches, in those extending toward the forehead. At other times the paralysis may be present only in the lower branches, which extend towards the nose and angle of the mouth.

Facial paralysis may come on suddenly without any premonitory symptoms, or it may be preceded by severe pain in the ear and corresponding side of the face, or by a twitching of the facial muscles.

Involvement of all of the cranial nerves except the olfactory and trothlear has been observed in extension of otitis media.

GRADENIGO'S SYNDROME.

This class of symptoms is more commonly a complication of acute otitis media, but it also occasionally precedes or accompanies mastoid infection.

Whitham[25] states that in Kollner's study of 250 cases of paralysis of the external rectus muscle of the eye, only 2 per cent were associated with otitis. This indicates the rarity of the syndrome. He also states that there is a mortality of about 18 per cent.

Gradenigo first described, in 1904, this symptom complex consisting of a diplopia due to abducens paralysis on the affected side, pain in the temporal and parietal regions of the affected side, with an acute purulent otitis media. His explanation of the syndrome was that there was spreading of the purulent infection from the antrum to the tip of the petrous portion of the temporal bone by way of the peritubal pneumatic spaces and the carotid canal, involving the abducens at the tip of the pyramid immediately after its exit from the dura. This involvement was thought to be due to an osteitis and pachymeningitis limited to the tip of the petrous bone. This would cause edema of the structures about the sixth nerve in Dorello's canal, with consequent pressure on the nerve, inducing paresis or paralysis. The petrous portion of the temporal bone, instead of being of a hard, bony character, may have become extensively pneumatized clear to the tip. This would permit of ready extension of infection from the middle ear or mastoid process. The pain in these cases is apparently due to the involvement of the Gasserian ganglion, which is in close proximity to the petrous tip.

Due to the fact that the Gasserian ganglion lies closer to a cell of the petrous tip than does the sixth nerve there may be involvement of the ganglion without involvement of the

nerve. This might give a trigeminal neuralgia without at any time an abducens paralysis.

Since this explanation was given several other explanations of the paralysis of the abducens have been advanced. At present there seems to be considerable difference of opinion and real doubt as to the cause of the diplopia.

The paralysis of the external rectus muscle usually appears from three to six weeks after the beginning of the otitis media. Operation is done only when there is a mastoiditis present or signs of a beginning meningitis.

Perkins,[26] after a study of many cases, says: "While recovery from the Gradenigo syndrome is possible without operative intervention, I believe that the syndrome itself, without apparent mastoid involvement, is sufficient justification for a mastoid operation at least."

EYE COMPLICATIONS.

There are a number of interesting conditions of the eye that may occur to complicate a middle ear or mastoid infection. While some of these disturbances are only symptoms of intracranial complications there are others that are in themselves real complications.

Emboli of pieces of clot or groups of bacteria may lodge in the arterioles of the eye and give multiple retinal hemorrhages, episcleritic nodules, iritis or possibly even metastatic choroiditis and panophthalmitis.

More often the eye is involved due to the diminished outlet of the retinal circulation secondary to complete thrombosis of the lateral sinus. This may give any degree of intraocular venous stasis, causing conditions varying from a pinkness of the optic nerve head or blurring of its borders to a distinct neuritic swelling with much engorged veins.

Fenton[27] states that in Uhthoff's series ophthalmic paralysis occurred in 12 per cent of sinus thrombosis cases. Abducens palsy was found in two-thirds of these, and partial oculomotor loss, usually of levator palpebræ, in about half. No fourth nerve cases were noted. Nystagmus was reported in 12 per cent of sinus thrombosis cases.

Accumulation of exudate, epidural abscesses and the like have occasionally been reported as extending directly along

the middle fossa to enter the orbit through the inferior orbital fissure. According to Fenton,[27] such cases have usually been in young children with numerous cartilaginous dehiscences and are also reported in persons with tuberculosis or diabetes. An orbital cellulitis of this type gives multiple palsies of the ocular muscles.

Prolonged epidural accumulation near the chiasm may occasionally bring about an accumulation of fluid around the optic nerve between the nerve and its dural envelope—a true perineuritis.

Ampulliform swelling of the optic nerve sheath and accumulation of fluid with interpenetration of the lymph spaces of the nerve is directly related to an increased intracranial pressure which may be secondary to a serous or septic meningitis or otitic brain abscess. Atrophy occurs when this pressure infiltration is long maintained.

It has been found that oculomotor nerve involvement is uncommon because of its high origin and protracted course. Direct injury will only occur upon the most extensive and complicated involvement of the petrous tip and cavernous sinus. Indirect muscular injury due to pressure is rare and transitory, but when seen is usually due to pressure forward from a central cerebellar abscess or inward from a temporosphenoidal abscess, toward the corpora quadrigemina or against the carotid artery. Such involvement usually affects only the levator palpebræ and the pupillary fibers giving ptosis and mydriasis (Fenton).

Crossed palsies involving the third nerve are rare possibilities with ear complications at the petrous tip or in the temporosphenoidal lobe.

Fourth nerve paralysis is very rare with ear complications but cases have been reported.

Sixth nerve involvement is the most frequent motor disturbance of the eye as an ear complication.

ERYSIPELAS.

Erysipelas occasionally occurs as a complication of suppurative otitis media and following the operation for mastoiditis. Hays[28] reports that in the New York Eye and Ear Infirmary, from January, 1912, to January, 1916, there were seventeen

cases of erysipelas. During that time there were between three and four thousand patients operated upon for mastoiditis, which brings the ratio down to less than four to a thousand, or about 0.4 per cent.

The usual explanation of the development of erysipelas following a mastoid operation is that the pus from the wound infects the adjoining skin. Hays is of the opinion that the organism causing the infection is present in the patient's hair and not due to the infection in the middle ear or mastoid cavity. He calls attention to the fact that in the majority of cases of erysipelas following a mastoid operation the erysipelas occurs in front of the ear and not behind it.

The complicating erysipelas probably usually occurs on the second or third day after operation, but may occur earlier and sometimes a week or more following the operation.

DERMATITIS OF THE EXTERNAL EAR.

In cases of chronic suppurative otitis media there may be any degree of involvement of the canal and pinna, from a mild irritation of the skin to intractable forms of dermatitis. There may result then a slight desquamative eczema or the condition may advance to a severe perichondritis with the formation of deep fissures and stenosis of the canal.

In the milder types the eczema may show in a loss of the epidermal layer of the skin, leaving an inflamed area with a slight exudate. There will be considerable itching and at times pain.

In the more severe cases there will be thickening of the skin, which may become so great as to close the meatus. The thickening in these cases appears to lie in the subcutaneous tissue and in the deeper layers of the skin.

In some cases there may be not only involvement of the skin and subcutaneous tissues but also of the perichondrium beneath. This results in a definite thickening of the perichondrium of the meatus and pinna, which, along with the thickened and adherent overlying skin, gives to the ear an appearance resembling the large flabby ears of the boxer. There is apparently no destruction of cartilage, but there is often a marked stenosis of the meatus with the skin thrown up in folds and the formation of fissures.

Dermatitis of the canal or pinna may commence within a few weeks of the first appearance of a suppurative otitis, or it may not appear for several years. The majority of cases appear within a year of the first evidence of a discharge.

The treatment of these cases is for the most part unsatisfactory. Long continued applications of medicinal agents may give considerable improvement, but few are really cured, and relapses frequently occur. In those cases in which a radical mastoid operation has been performed there has sometimes occurred an acute perichondritis, which would be likely to leave a shrunken and deformed ear. There is also the danger of the formation of a complete stenosis of the meatus.

PAROTITIS.

On account of the close relationship between the ear and the parotid gland infection may easily extend to the gland by continuity or by lymphatic metastasis. The break in the continuity of the cartilaginous portion of the auditory canal by the fissures of Santorini make an extension more likely to occur.

Involvement of the parotid gland must be distinguished from swelling of the pretragal lymphatic glands, which are one or two in number, located just in front of the pinna, and sometimes are within the substance of the parotid gland. There must also be differentiated an abscess under the temporal muscle due to infection in the zygomatic cells.

FOCAL INFECTION.

The infected mastoid process may act as a focus of infection in the same way as the teeth, tonsils or sinuses. In cases of chronic purulent otitis media with pus lying in the antrum and adjacent mastoid cells there may be poor drainage of the pus into the tympanic cavity. This would be apt to result in absorption of toxic material into the system, with the same effects that are more commonly produced by a focus of infection in the teeth or tonsils.

Many of the chronic mastoid infections give no symptoms referable to the mastoid itself and may only give some purulent discharge from the ear, often at rather long intervals. When the mastoid cortex is thick or when there is a relatively large amount of diploe structure and little pneumatization of

the mastoid there may be no local evidence whatever of any infection in the mastoid cells.

Some of the conditions that have developed secondary to mastoid infection are neuritis of the sciatic nerve, acute articular rheumatism, acute nephritis, aggravation of certain psychoses, anemia, bronchitis and septicemia with metastatic abscesses.

Absorption of toxic material in the middle ear and mastoid quite often affects the immediately adjacent structures to cause alarming and serious conditions, such as a facial paralysis or a labyrinthitis.

GASTROINTESTINAL DISTURBANCES.

Within the last few years there has been frequently brought to the attention of otologists the relationship existing between middle ear infections and gastrointestinal disturbances in infants. Whether these disturbances are a complication of the otitis or whether the mastoiditis is a complication of the intestinal infection has not been definitely decided. There remains, however, the fact that an antrotomy on these infants causes a subsidence of the intestinal disturbances in many cases.

The character of these disturbances in infants seems to be at first a failure to gain normally in weight, which goes on, in severe cases, to marked loss of weight, vomiting, diarrhea, anhydremia, extreme toxicity and a septic temperature with leucocytosis. These symptoms have been thought by some to be due to a toxemia of infectious origin, the location of this infection being, in the majority of instances, in the ears.

The condition of the ears in cases with these symptoms varies somewhat. At times there is found a frank purulent otitis media, very often of both ears. In other cases very little of a pathologic significance can be seen. The drum is apt to show a change of color to a dirty gray opaque appearance. In some cases it is red and bulging. The most constant finding is a sagging of the superior or posterior superior canal wall just external to the tympanic membrane.

In some of these cases adequate drainage can be obtained by an incision of the upper portion of the drum and through the membranous floor of the antrum or upper wall of the external auditory canal. It is often necessary to incise this region repeat-

edly. In other cases, especially in those with severe gastro-intestinal symptoms, it is necessary to open both mastoid antra. These antra usually contain frank pus or a thick, ropy, mucoid fluid, and the embryonal tissue shows varying degrees of destruction. It has been found that marked changes in the bone have not occurred.

CERVICAL ABSCESSES.

Cervical abscesses occur rather frequently with mastoiditis and tend to complicate the mastoid operation somewhat and make the prognosis more unfavorable.

Kelly[29] has made a careful study of the various types of cervical abscesses and their extension into certain muscle groups. It was found that variations in the thickness of the mastoid cortex has much to do with the development of these abscesses. In the type of mastoid with an outer cortex quite uniformly thick and the tip and inner cortex very thin there is a predisposition to the formation of a fistulous opening on the mesial surface of the mastoid tip in the region of the digastric groove, causing a Bezold's abscess. Fistulous openings through different points of the mastoid cortex cause pus to burrow into certain muscle groups or fascias of the neck. This pus may extend forward to the pleural apex and clavicle or behind to the scapulæ and vertebræ of the neck. It may extend between the superficial and deep throat and neck muscles to form abscesses in the neighborhood of the pharynx which may perforate into the pharynx, or the pus may cross the midline of the neck to form abscesses in the muscle groups of the opposite side.

Fistulæ that perforate the mastoid tip externally give rise to sternomastoid muscle infiltration. Those occurring more externally on the inner cortex dispose, because of the backward curve of the splenius and longissimus capitus muscle, to postoccipital or postvertebral abscesses, while deeply located fistulæ, found more mesially on the inner cortex, perforate the digastric muscles to become a prevertebral or retropharyngeal abscess.

Abscess in the retropharynx may also occur by an invasion of the suppurative process into the tip of the petrous portion of the temporal bone, breaking through the lower surface of

the bone to reach the posterior part of the pharynx. Extension of this kind involves also the sixth nerve and the Gasserian ganglion, giving the characteristic picture of Gradenigo's syndrome.

Another possible route for pus to travel into the retropharyngeal region is along the eustachian tube from the middle ear, under the mucous membrane or periosteum and perichondrium, to the postnasal space.

One of the more rare locations for such types of abscesses is in the pterygomaxillary or infratemporal fossa. Possible routes of infection to this region are from diseased cells in the digastric groove of the mastoid process, infected cells in the petrous portion of the temporal bone, and through an anatomic defect in which the crista tympanica and tegmen tympani fail to unite, thus leaving a communication between the tympanic cavity and the pterygomaxillary fossa by means of the Gasserian fissure.[30]

PROTEIN FEVER.

Hays[31] mentions a condition known as protein fever which may seem more an unusual symptom following mastoid operation than a real complication. This condition appears following a mastoid operation as an irregular temperature, at times very high, which cannot be accounted for and which subsides after a few days or weeks. It has been thought to be due to absorption into the system of the proteins of some retained bacteria but this seems a questionable supposition.

A rise of temperature after a mastoid operation, with or without a chill, may be due to the absorption of infected material or the toxins of the infection in the temporal bone. This especially would be liable to occur if the sigmoid sinus had been uncovered during the operation, the absorption taking place into the sinus through the vasa vasorum.

DEAFNESS.

Deafness following mastoid infections is due not to the mastoid involvement but to the preliminary disease and often destruction of tissue in the middle ear.

After the infection of the middle ear has subsided, the perforation in the drum has closed, and the mastoid wound has

entirely healed, a catarrhal condition may begin in the middle ear, become chronic and give a slowly increasing deafness and tinnitus.

Progressive impairment of hearing may result also from adhesive processes, which develop quite commonly after middle ear suppuration. The exudate becomes organized and adhesive bands form, binding the ossicles to each other or to the walls of the tympanic cavity, or forming bands between the drum and the inner tympanic wall.

In some cases, especially those accompanying infectious fevers, such as scarlet fever and measles, there is a partial or complete deafness due to extensive destruction of tissue within the ear. The tympanic membrane and ossicles may be partially or entirely destroyed, and there may be necrosis of the promontory or perforation of the round window leading to the labyrinth.

It has been found that the bone of the two larger ossicles is very resistant to the attack of caries. They may lie embedded in pus, granulation masses or cholesteatoma for years without any apparent change. However, though they seem to be intact, they may not be fulfilling their function of transmitting sound vibrations. Caries most commonly attacks the incus at the extreme tip of the long process, and destruction at this point, even if only small in amount, will break the continuity of the ossicular chain. Again, any softening of the structures forming the joint between the malleus and incus will interrupt sound transmission and result in deafness.

MASTOID FISTULA THROUGH BONY WALL OF MEATUS.

Dr. Watkyn-Thomas has reported three cases showing an unusual complication of mastoiditis, in which there was a fistula through the posterior bony wall of the external auditory meatus. This allowed pus from the mastoid to extend through the wall of the meatus underneath the skin covering of the canal, giving the appearance of a furuncle. In cases of mild mastoiditis with no tenderness and little or no elevation of temperature cases of this kind might easily be mistaken for and treated as conditions of furunculosis of the canal.

RECURRENCE OF MASTOID INFECTION.

A recurrence of mastoid infection, in the great majority of cases, is due to the incomplete exenteration of the mastoid cells at the time of the original operation. When the pneumatic structure is entirely obliterated prompt and complete closure of the mastoid wound takes place and the purulent discharge from the ear ceases, usually within a week or two. When there is a persistence of the discharge from the ear or when the mastoid wound fails to close, leaving a discharging sinus behind the ear, it usually means that some diseased mastoid cells have not been obliterated.

The cells that are most commonly overlooked are probably those which lie just above the knee of the lateral sinus between this structure and the roof the tympanum. The cells located at the root of the zygoma are next most frequently overlooked, and thirdly are the deep cells in the lower portion of the mastoid either in front or behind the lateral sinus.

The chief reason that a complete exenteration of the mastoid cells has not been done is because the operation has been performed too soon. When suppuration has developed within the mastoid there is a softening or breaking down of the intercellular bony tissue. It is then much easier to do a complete obliteration of the mastoid cellular area than it is earlier, when the process is still in the active inflammatory stage. In this stage it is much more difficult to find and eradicate every contaminated cell, and if any are left behind they may not drain sufficiently to recover. In that case there may be a continuation of the discharge, or there may be a breaking out of the infection at a later period, necessitating a second operation.

In another class of cases in which the mastoid operation has been complete a recurrence of a mastoid infection may occur because there has not been a closing off between the mastoid cavity and the attic. In these cases a second infection of the middle ear may cause pus to extend back into the mastoid cavity which has not become filled with granulations. This would result in a secondary mastoid abscess.

In the consideration of this subject that takes in so many conditions of importance only the briefest mention has been made of many of them. Only some of the general features of

each complication, with certain statistical data, have been recorded. Emphasis has been laid on the factors that tend to produce these complications and on the method of extension from the original source of infection, with practically no mention of the symptomatology or the interesting features of differential diagnosis. It is hoped that this review of such an extensive subject, so lacking in the clinical features that tend to lend interest to a paper, may still find some value if only as a source of reference for data on the occurrence of complications and the mode of extension of infections from the middle ear and mastoid process.

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XVIII.

INFANTILE MASTOIDITIS WITH GASTROINTESTINAL SYMPTOMS: REPORT OF TWO CASES.*

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St. Louis.

The close association of infantile mastoiditis with severe gastrointestinal symptoms is now well recognized. Numerous reports in the literature in recent years have shown the value of mastoid antrotomy in relieving the gastrointestinal symptoms. Attention has been so centered upon the operative procedure that one may be led to assume that no further treatment is necessary other than proper feeding, when, as a matter of fact, treatment directed to a restoration of a normal chemistry of the body is of as great importance as the operation itself. The following cases are reported because they illustrated the importance of the measures mentioned.

Case 18.—Salvatore R., referred to hospital by Dr. L. A. Robertson on October 22, 1927, age 2 months on admission. Baby was normal at birth, breast fed a short time, and then given sweetened condensed milk, which was followed by diarrhea and loss of weight. He was put on lactic acid milk by Dr. Robertson and did well until just before admission to hospital. At the time of admission he was having from 15 to 17 loose, watery stools per day, and vomiting most of his feedings. Paregoric, powdered protein milk and other measures failed to relieve the vomiting and diarrhea.

Examination showed the baby dry, gray and undernourished. The pharynx was inflamed. The temperature was fluctuating from 37 to 38 C. The ear drums, which were red, thickened and bulging, were incised, with the escape of considerable serous material. Dr. Marriott's diagnosis was diarrhea due to parenteral infection (otitis media with probable mastoiditis).

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*From the Department of Oto-Laryngology, Washington University School of Medicine, and the St. Louis Children's Hospital.

Supporting fluid was given intraperitoneally, as Ringer's solution, as shown on reproduced chart 1. On October 24th, 90 cc. of the father's blood was given intravenously. The number and fluid nature of the stools continued. On October 25th, air hunger developed, blood was taken for chemical study and glucose given intravenously. Five per cent soda bicarbonate solution was given intravenously as shown in chart 3, and from this point glucose solution and fluid by mouth were depended on for fluid support and diuresis, for the reasons set out by Dr. Hartmann in the following note:

October 26, 1927. "The marked persistent acidosis probably resulted as follows: Infection in the mastoids caused diarrhea and vomiting, with resultant anhydremia. This was relieved by frequent administrations of Ringer's solution. Parenteral infection continued, diarrhea continued, and water was lost from the baby by way of the gastrointestinal tract to a relatively much greater extent than salt was lost from the body. Some BCl and $BHCO_3$ must undoubtedly be present in vomitus and stools. This loss, particularly of $BHCO_3$, coupled with the gain of NaCl, KCl and $CaCl_2$ in the Ringer solution would account for the low CO_2 content of the blood with the extremely high NaCl content. The retention of chlorid is probably largely due to the oliguria, which also causes retention of N. P. N. and phosphate. The indications, besides antrotomy, are to relieve acidosis by careful intravenous administration of $NaHCO_3$, to force water by mouth and to give glucose intravenously to produce diuresis so that chlorid may be excreted, and to discontinue all chlorid administrations."

October 26, 1927. Operative Note.—Using novocain-adrenalin beneath the skin and periosteum, the cortex was removed over the mastoid antra in the usual way, revealing antra of relatively enormous size, half filled with yellowish mucopus. Both antra measured irregularly 15 x 9 x 7 mm., which is the average dimensions given by anatomists (Piersol, etc.) as the adult size. (It is stated that the cell at birth should be the size of a small pea, or about 5 mm. in diameter.) Sterile saline introduced into the antra of this little patient and withdrawn into a syringe measured a little more than 1 cc. each. No structure was touched except the cortex, and care was taken not to curette any of the mucosa. Enough cortex was removed

to expose the cavity completely, and then the simple gauze dressing was applied.

Culture from the antral secretion showed at the time of operation a luxuriant growth of bacillus coli communis and staphylococcus.

At the daily dressing the antra were treated with 1 per cent mercurochrome and on the third postoperative day, October 29, the colon bacillus did not occur in the culture but staphylococci were present. At about this time toxic symptoms seemed to have entirely disappeared. There were no diarrheal stools after the 28th, becoming pasty, then formed. The baby rapidly improved with gain in weight, without parenteral fluid administration, as shown in chart 1 and 2, the dotted line indicating the weight curve.

Case 28.—Donald M., age 6 weeks. This child was a difficult feeder from birth, never seeming very strong and having a peculiar weak cry. The stools were always loose. The five weeks previous to admission to hospital was jaundiced. On the day of admission child had a vomiting spell; following this he became very gray in color.

October 14, 1928. Examination showed a cyanotic dry baby, hypertonic in every respect. The abdomen was markedly distended. No meningeal signs, any of which might suggest a spastic state to be associated with birth injury. The urine showed a strong trace of albumen, numerous cylindroids, granular casts and pus casts. W. B. C., 28,400; R. B. C., 3,950,000. Right ear drum red, bulging, and incision recovered considerable serous fluid.

October 14, 1928. Two hundred and fifty cc. saline was given subcutaneously. Large visible peristalsis evident.

October 15, 1928. (Note by Dr. Marriott).—This child is picture of dehydration and intoxication, skin dry and gray. The respirations are deep and pauseless. Much undernourished. Waves of gastric and intestinal peristalsis. No meningeal signs. The left drum is fiery red and there is a seropurulent discharge from the right. I believe this child has high chlorids in the blood and low bicarbonate. It requires dextrose and bicarbonates. Blood chemistry to be done. Diet, skim lactic and dextrin. May need mastoid antrotomy.

October 15, 1928. CO_2 15; P. H. 7.14; NaCl 831; protein 7.20. The patient was given 100 cc. of 10 per cent dextrose and 1½ grams of sodium bicarbonate intrasinus.

January 15, 1928. (Dr. Costen).—Under local anesthetic, ½ per cent novocain with adrenalin, both mastoid antra were opened, revealing large irregular spaces, lined with yellowish pyogenic membrane. There was present a moderate amount of mucopus, more in the right side. The volume of each (measured) was .8 cc.

October 17, 1928. Baby improved after a sharp postoperative rise in temperature, when for one day the prognosis looked very doubtful. Cultures from the antra showed no growth. Stools less frequent, temperature lower, color improved.

From the second postoperative day, the child gained weight at a phenomenal rate, stools became less frequent and the temperature subsided. At the various times shown in chart 3, glucose was used for fluid support. This was not necessary after the second day. Following this, it gained 700 grams in one week. It was discharged on October 22, 1928, and observed at intervals for two months. It has had no further trouble and was growing at the normal rate.

	Blood																Therapy				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)	(21)
		Sodium Chloride		Carbon Dioxide Content		Protein		Inorganic Phosphate		Lactic Acid				Nonprotein Nitrogen							
Case 18 Date	pH	Mg. per cent	BA† Equiv. Cc. N/10	Per Cent by Volume	BA Equiv. Cc. N/10	Per Cent	BA Equiv. Cc. N/10	Mg. per cent	BA Equiv. Cc. N/10	Mg. per cent	BA Equiv. Cc. N/10	Total Acid Determined, Cc. N/10	Total Base, Cc. N/10	Mg. per cent	BA Equiv. Cc. N/10	Total BA Equiv. Determined, Cc. N/10	Freezing Point — C.	Date	Ringer's Solution, Cc.	Dextrose, 10-20% Cc.	Blood, Cc.	Sodium Bicarbonate 5% Cc.
10/25/27	7.13	802	137	17.1	7.2	8.92	14.1	7.9	4.6	68.0	7.5	170	162	60.0	10.7	181	---	10/22 to 10/26	1,600	100	90	100
10/26/27	7.30	714	122	23.5	10.0	7.20	12.2	6.9	4.0	64.2	7.1	155	157	61.0	10.9	168	---	10/26/27	----	100	--	50
10/27/27	7.27	667	114	24.5	10.4	7.42	12.4	7.7	4.5	37.8	4.2	146	155	85.0	15.2	170	550	10/27	---	100	--	50
10/28/27	7.30*	591	101	31.0	13.2	6.77	11.5	---	4.5*	---	3.6*	126	143*	76.0	13.5	157*	---	10/28	---	50	--	50
11/ 4/27	7.28	645	110	29.0	12.3	7.20	12.2	8.0	4.6	26.5	3.0	142	136	50.0	8.9	151						
11/11/27	7.39	591	101	51.5	21.8	6.34	11.2	---	4.0*	---	2.5*	141	137	42.0	7.5	140						
Case 28:																						
10/15/28	7.11	831	---	15.0	---	7.20	---	---	--	---	--	---	---	---	---	---		10/14/28	250	---	--	---
																		10/15/28	---	100	--	30
																		10/16/28	---	175	--	50

*Assumed values.
†Acid-base equivalent.

Chart 3. Record of blood determinations before and after the fluid support was limited to dextrose intravenously and water by mouth.

Results of the blood chemical examination in this case are reproduced here, with the permission of Dr. Hartmann, for the sake of emphasis on the accuracy with which the supporting treatment may be applied.

A full discussion of the blood chemistry findings and of the rationale of treatment adopted is beyond the limits of the present paper. For a fuller discussion the reader is referred to the paper of Hartmann.[8] The striking result obtained in this case of an infant apparently moribund demonstrates in a graphic manner the value of the practical application of the theoretical principles involved—i. e., using sodium bicarbonate in amounts equivalent to about one-fourth the normal content of the body fluids, with water and dextrose solution for fluid support.

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722 Beaumont Medical Building.

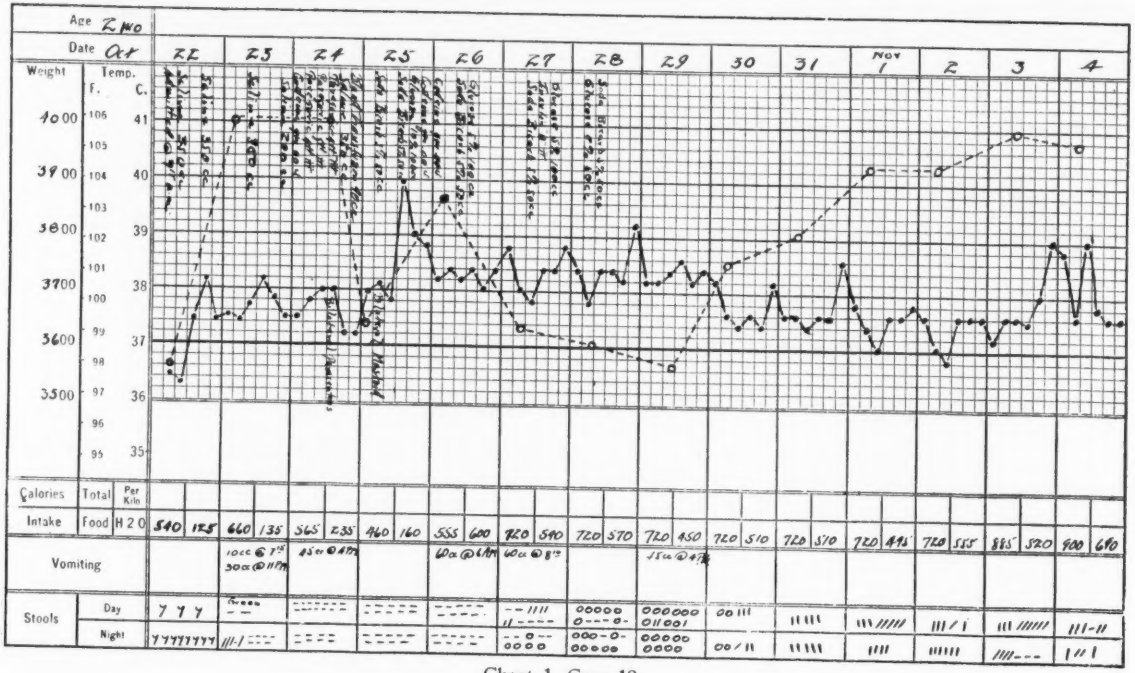

Chart 1, Case 18.

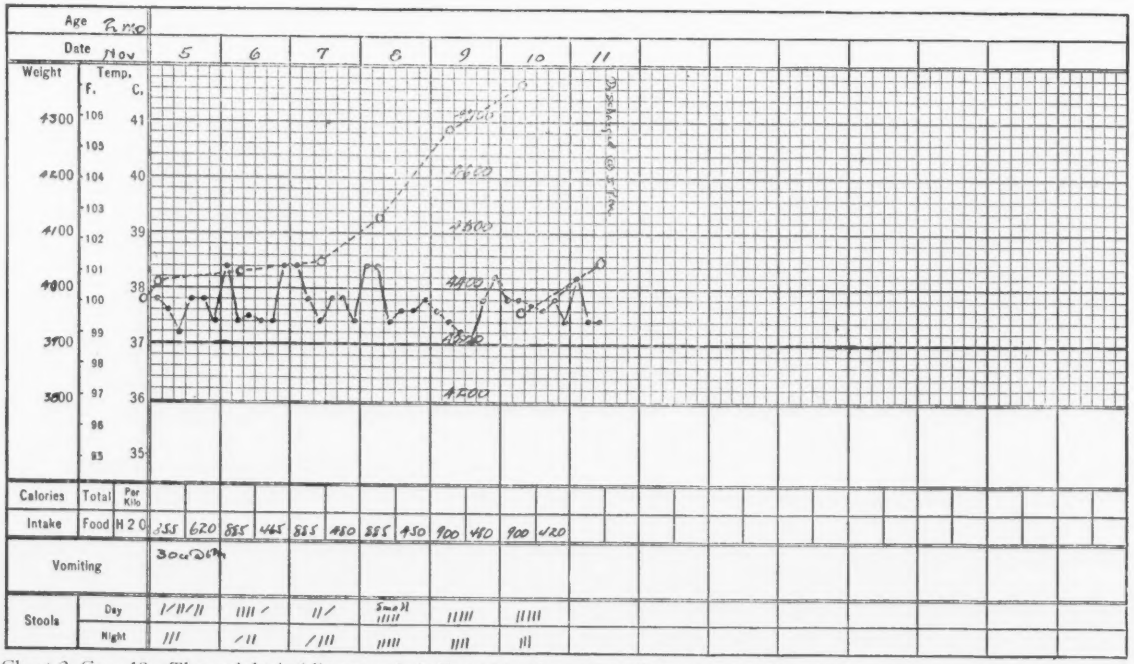

Chart 2, Case 18. The weight in kilograms is indicated by the broken line; the solid line is temperature; watery stools are short horizontal lines, and normal stools are short vertical lines; pasty stools are small circles.

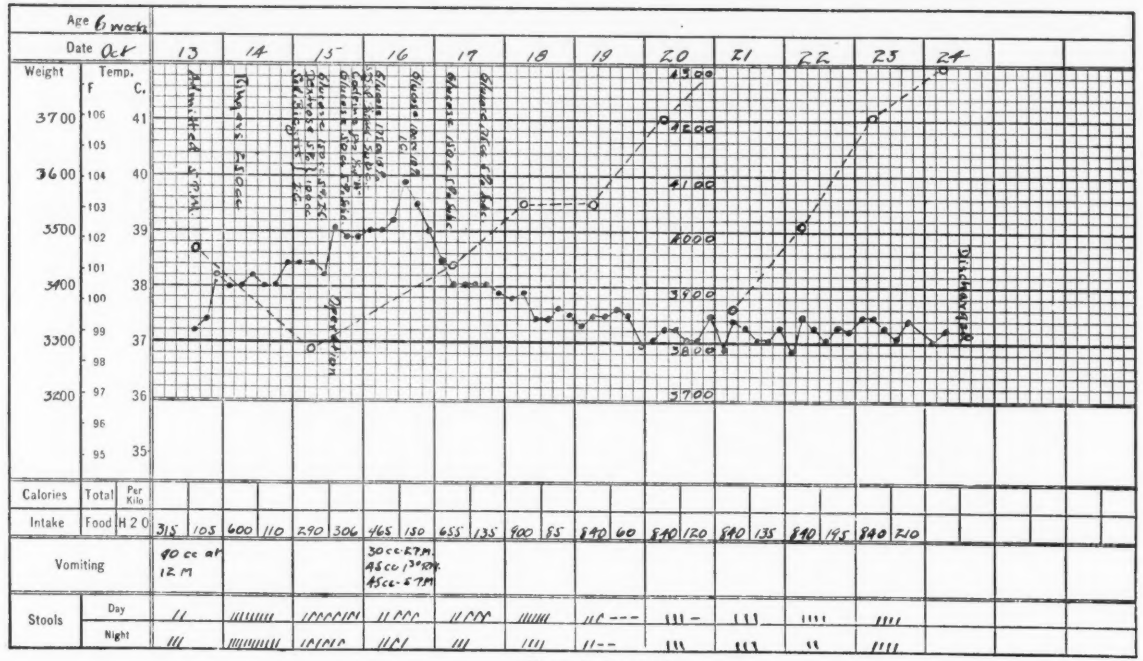

Chart 3, Case 28.

XIX.

NONCOALESCENT MASTOIDITIS WITH A REPORT OF FOUR CASES.

By Sam B. Marks, M. D.,

Lexington, Ky.

During the last year, 1928, it has been my lot to encounter four cases of mastoid disease of high virulence and toxicity. Each of these cases has shown clinical phenomena of so widely different character, one from the other, that to me they form a group, the study of which has been of great value. I have classed them in the noncoalescent group of Dr. S. J. Kopetzky of New York, whose simple, concise and comprehensive classification of acute mastoid disease into coalescent mastoiditis and noncoalescent mastoiditis, being based as it is upon the anatomic and pathologic findings, is, to the best of my knowledge, universally accepted as the most logical. These cases were all in private practice, most carefully watched by the internist, both before and following operation, and all were carefully treated with frequent instillations and irrigations of warmed 2 per cent mercurochrome every two to four hours as the necessity arose, to keep the canal and the middle ear as free as possible of any obstructive discharge, from the inception of the middle ear infection.

It is here that I wish to gratefully acknowledge my appreciation to all other physicians associated with me in the conduct of these cases, as their advice and cooperation were of the greatest assistance.

Case 1.—A well nourished white boy, eight years old, with no previous history of ear trouble. I had removed his adenoids and tonsils two years previously. He was seen at the office by my associate on January 25, 1928, with an earache on the left side. The drum membrane was red and dull, but not bulging, and upon incision a small amount of thin fluid was obtained.

I saw the patient the next day with no pain, a free opening in the lower posterior quadrant and only a little moisture. The

following night I saw him with temperature of 103 and severe pain in the same ear with a free discharge of thin mucopus. The membrane was edematous above and very red, and was again incised under chloroform anesthesia. The following day he was comfortable with little fever and a free discharge, but that night the pain and temperature returned and the membrane was more edematous with profuse discharge and pulsation in the middle ear, so again he was chloroformed and the incision investigated but found freely open. Dr. John McGinnis, the family doctor, also saw him and reported negative physical findings, as were his nose and throat and other ear. The urine was normal with no pus or bacteria. At no time was there tenderness or other sign of mastoid involvement. The next morning he was bright and comfortable. That evening his temperature was again high, but he had no pain in the ear or signs of mastoid involvement. He was removed to the Good Samaritan Hospital, where the urine was again negative, but his leucocytes were 18,800 with 94 per cent polymorphonuclear cells, so the left mastoid was freely exenterated; the sinus plate was being exposed but found healthy. The antrum and tip cells, covered by an unusually thick layer of bone, contained dark mucopus and edematous discolored membrane; the rest of the bone was softer than normal, with many small cells containing a pinkish membrane but no pus. The roof of the aditus was likewise soft, but no pus was found in the zygomatic region. For forty-eight hours the temperature ranged between 103.6 and 100.6, with a pulse of 103 to 120, and respirations 24, but other than a normal irritability and tenderness of upper part of the sternomastoid muscle he was in a normal state. The urine was negative as was his chest. The following day his condition was normal with highest temperature 100.8.

The fourth day his temperature rose to 102.2 and a few pus cells and many bacteria were found in his urine. Leucocytes were 10,000 with a polymorphonuclear percentage of 70. A culture from the urine showed colon bacilli. For the following four days his temperature ranged from 103.2 to 105.2, and at which time there was a marked bacterial shower in the urine, this being on the eighth day. I then found he was inadvertently getting both an alkalin water and urotropin, and upon stopping the alkalin water his temperature receded, ranging

between 100.4 and 98, with a slowly clearing urine. During this period of five days the leucocytes were 18,000, with polymorphonuclear count of 74 per cent; Widal negative, chest negative, blood culture from arm negative, and during the whole time the boy felt fine and was propped up in bed drawing pictures and amusing himself. The mastoid wound was progressing nicely and rapidly healing.

The thirteenth day his temperature again rose to 104.2 and in four hours was 99.6 and again in four hours to 103.6, with a recession to 99.6 in four hours, with only a few cells in urine and no bacteria. At this time the metatarsophalangeal joint of the right great toe was red and swollen and tender but was much better the next day, whereas his temperature again rose to 103 in eight hours. I then took a blood culture, on the fourteenth day, from the right internal jugular vein, after the technic of Dr. Rueben Ottenberg, and found in thirty-six hours an enormous growth of hemolytic streptococci on a glucose-agar plate. I failed to get blood from the left internal jugular.

The patient was then prepared for an operation upon the lateral sinus of the operated side, which proceeded as follows: The left internal jugular vein was rapidly exposed for its upper one-half and found to be nearly empty of blood with a thick wall and very much narrowed lumen, looking more like an artery than a vein. It was doubly ligated and divided at site of facial vein, which was also ligated; the wound was closed by closing the cervical fascia and skin with catgut, leaving a rubber tissue drain from the site of the ligation to protrude at the lower wound angle.

The mastoid wound was then opened to its full width extended posteriorly, the sinus ridge more fully exposed and the plate removed from the knee of the sinus to the jugular bulb. The sinus was not visibly pulsating but was to touch; its wall was thickened and of a pale yellow color, and upon opening the sinus it was found filled with thin yellow pus, being blocked both above and below by a dense, well organized clot which extended posteriorly fully half way to the torcular, the wall being covered with a pyogenic membrane at the posterior end of the thrombus where the clot was fresh and contained thick yellow pus. After free bleeding was allowed the sinus was plugged with iodoform gauze and the lower end was investi-

gated, finding only the old clot, which was removed but no free bleeding obtained. The sinus wall was then excised, after putting in the lower iodoform plug. The wound was packed with iodoform gauze, only the posterior extension of the wound being sutured over the gauze.

The next two days the patient was irritable and cross with a temperature of 98.8 to 101.6 and a clear urine. From this time the convalescence was uneventful, the last packing being removed on the twelfth day and the patient going home on the sixteenth day with complete healing two weeks later.

Case 2.—A well nourished white girl, aged six years, whom I saw with Dr. Tom Marks on March 29, 1928, with the following history: Two weeks before she had had hemorrhagic measles, but had progressed normally until three days ago, when her temperature suddenly rose to 104 with nothing to account for it. Chest, urine, nose, throat and ears negative. Two days later she complained of her ears and became quite deaf, and the drum membranes were both found thickened, red and bulging. I opened both ears freely the same afternoon under chloroform anesthesia, and the following day she was much better in all respects with a marked improvement in hearing and a profuse thin discharge from each ear.

The next day her temperature again rose, the ears still profusely discharging, but no pulsation and no signs of mastoid involvement. Dr. Tom Marks, her physician, found distinct bronchial breathing over the middle lobe of the right lung and we thought this was the trouble. There also had developed soreness in the right shoulder but no redness or swelling, of which nothing developed, and swelling and tenderness at first rib and sternal joint on the right side, which later suppurated. The next day she was so ill she was removed to the Good Samaritan Hospital for further study. Both mastoids were X-rayed but showed very slight if any changes. The chest showed light feathering of the middle lobe with no positive physical signs. The urine was negative. Leucocytes 21,550 with 85 per cent polymorphonuclears. Temperature 104.2, pulse 132 and respirations 40, and profuse thin mucopus from both ears.

Later in the day she was prepared for operation and both mastoids were opened, the left first, as the discharge in this

ear was pulsating, but not in the right, with the following findings: The antrum and a few neighboring cells were found filled with the same character of pus as the ear and a thickened granulomatous lining membrane, but the sinus plate was intact with solid bone and was not opened. The right mastoid was opened, but less extensively so, with virtually the same findings.

After dressing the wounds blood was taken from the right internal jugular, again by the Ottenberg technic, but I was unable to enter the left vein. This culture was strongly positive for hemolytic streptococci in twenty-four hours.

A spinal puncture, made under the anesthetic before the operation, showed a clear fluid under moderate pressure with a normal cell count and no growth.

The patient was desperately ill for the next five days, with a temperature of 103 to 106, pulse 130 to 150 and respirations 30 to 40, but took orange juice and water copiously and was given digifortis 15 m. every four hours.

On the fifth day I opened, under novocain, an abscess external to the right sternomastoid muscle, just above the clavicle, coming from the first rib and sternal joint and had to reopen over the joint two days later; both abscesses were deeply seated with the sternum uncovered posteriorly between the first and second ribs. Following this she improved enough, with temperature 101 to 104, pulse 135 to 145 and respirations 40 to 48, to submit to a chest examination and X-ray on the seventh day, which showed right lower and middle lobes densely consolidated with many coarse moist pleuritic râles. Dr. Tom Marks aspirated this side but obtained no pus. Here she began to slowly show slight continuous improvement, but as her pulse became quite irregular the digitalis was stopped for a few days. The tenth and eleventh nights she had pseudocrises and almost died, her temperature receding to 101 and 99.6, respectively, and extreme prostration occurring.

The fifteenth day she was again X-rayed and a small empyema was found on the right side, from which 130 cc. of dark thin positive streptococcic fluid was withdrawn, and again upon the seventeenth day 160 cc. were removed, the fluid now

being purulent. Her condition showed marked improvement after each aspiration, and upon the nineteenth day Dr. C. A. Vance drained the empyema between the seventh and eighth ribs under novocain and put in a valve drainage.

After two days of considerable disturbance she began to really get well, the mastoid wounds healing on the thirty-first day and the sternal abscess a few days later. Following this nothing else happened except the incision and drainage of abscesses of the left forearm and right buttock, on the forty-ninth day, by Dr. Vance, under chloroform. She made an uneventful recovery and all wounds healed.

Case 3.—A white girl, nine years old, thin, but in good health and no former history of significance, presented the following history: She contracted a severe attack of measles ten days ago, but was convalescing satisfactorily until I saw her on May 15, 1928, with a severe left sided earache of six hours' duration and a temperature of 100. The membrane was dark and bulging, and when freely incised, with chloroform anesthesia, a small amount of thin fluid and very free bleeding was obtained. For two days she progressed favorably, with moderate discharge, no pain and very little fever. She then became quite ill with temperature of 104, pulse 120 to 130 and constant vomiting of all food, medication and frequently water. Her urine showed many pus cells, colon bacilli and four plus reaction for acetone. When she showed no improvement in two days, with treatment of the above findings, I was again asked to see her, and examination revealed the following: General examination by Dr. J. P. Warren, her physician, showed nothing except a persistence of the urinary findings and a marked restlessness and irritability, with no increased reflexes. Temperature was 106, pulse 140 and the left ear discharging profusely a thin mucopus. There were no signs of mastoid involvement except moderate tenderness over the mastoid. The right ear was negative, as were her nose and throat. Leucocytes 21,200, polymorphonuclears 81 per cent. A blood culture taken at this time was negative. She was taken to the Good Samaritan Hospital a few hours later and the left mastoid operated upon with the following findings: A large cellular mastoid was found with marked softening in the antrum, zygomatic and postsinus cells, all of which contained dark fluid

and dark swollen membrane. The tip and other cells were less affected but contained the same dark fluid and membrane. The sinus plate was healthy and sound.

A spinal puncture, done under the anesthetic at start of operation, though the fluid was under pressure, showed negative findings. The convalescence was normal with a sudden subsidence of all symptoms, although she did not realize she had been operated upon for three days, and a right middle ear suppuration had to be opened on the same day.

Case 4.—A well nourished white boy, seven years old, whom I saw in consultation with another otologist, Dr. G. B. Brown, with the following history: Past history of no significance. On December 12, 1928, he had a mild left sided earache with a temperature of 99 for an hour or two. The next day there was a return of the pain, which became severe, and his temperature rose to 102. A full myringotomy was done, followed for four days by a moderate thin mucopurulent discharge, the membrane then healing. Two days later the drum membrane was found dull red and bulging, with a return of pain and fever, and was reopened with a discharge of thick mucopus. Two days later Dr. Brown and Dr. Tom Marks asked me to see him. Although he was free from pain the discharge persisted with a temperature of 102.5 and pulse of 100. His condition at this time was good, with negative general findings, except slight irritability and loss of appetite. The urine was and remained negative, leucocytes 22,000, with a polymorphonuclear count of 78 per cent. An X-ray showed his mastoids normal. A moderate amount of thick mucopus was escaping from a good opening in the lower two-thirds of the posterior quadrant of the membrane, with slight edema of the flaccid membrane; there was no edema of the canal wall and very slight, if any, tenderness over the mastoid bone and none over the jugular vein. The right ear, nose and throat were negative. At my suggestion the myringotomy was extended above into the canal wall, but his condition remained unchanged, and three days later his ear again became closed and his temperature rose to 104, necessitating another myringotomy. The following day he was no better, and over the telephone I advised that a complete blood count and blood culture be made, which showed the following: 4,900,000 red cells, hemo-

globin 80 per cent, leucocytes in the morning 14,000, the polymorphonuclears being 82 per cent, against 11,200 and 78 per cent in the afternoon. The blood culture was reported positive for hemolytic streptococci in the thirty-six hours. For the past three days his temperature had shown rises and falls every twenty-four hours. Upon the blood culture report he was removed to the Good Samaritan Hosptial, and I was asked to operate upon him. At this time his temperature was 103, leucocytes 8,300 and polymorphonuclears 81 per cent. There were no positive signs of mastoid involvement and his general condition was good. The operation revealed a dense vascular mastoid with a very thick cortex and all cells small with no destruction, but bloody fluid and an edematous membrane. The antrum was small and contained mucopus, but the tip cells, of fair size, contained only serum and thickened membrane. The sinus plate was clean and intact, the posterior sinus cells being the same character as the others. The zygomatic cells were clean. The day following his condition was good but his temperature again rose to 104.5 for four hours and then receded to 100, where it remained during the next twenty-four hours, at which time he was given 200 cc. of citrated blood from his mother. A blood culture taken at this time was positive in thirty-six hours, but otherwise his condition was satisfactory, there being no further temperature rise except a moderately severe transfusion reaction, and his convalescence was uninterrupted. A blood culture taken 48 hours after the transfusion showed only a few colonies after 72 hours. The leucocytes at this time were 9,500. This case was given, at the suggestion of Dr. Tom Marks, a daily dose, for seven days, of one-half of a cubic centimeter of immunigen, a filtrate of a combined culture of the streptoccocus, prepared by Parke, Davis and Company. The first was given the day before the operation.

CONCLUSIONS.

1. Case 1 should have been diagnosed as one of sinus thrombosis at an earlier period, but we were misled by the boy's excellent condition, the frequent recrudescences of his temperature curve, the last one being of five days' duration, and by the continued positive urinary findings.

2. Case 2 was most remarkable in her recovery, due in large measure to her ability to take large amounts of fluids and to her inherent placidity and stoicism, as at no time did she admit feeling other than fine and slept for long intervals and was quite free from that consuming restlessness so common in such cases. Transfusion was not done because she was thought too ill to withstand the shock and reaction should any occur.

3. Case 3 was unquestionably in a state of an early serous meningitis, probably well localized, which would, I believe, have rapidly become septic had operative interference not been immediately resorted to. The presence of both pyelitis and an active acidosis in this case were factors of very great interest.

4. Case 4, showing an actively positive blood culture with no complications resulting, and the low leucocyte count for 48 hours preceding operation, was of great interest. The one was due to a direct blood infection from the mastoid, with no sinus or other involvement, and we have no explanation of the other, as the patient did not show evidence of an overwhelming infection. Cognizance of the use of immunigen as a factor contributing to both the subsidence of the blood infection and the presence of the low leucocyte count cannot, we believe, be disregarded.

5. This small group of cases serves its usefulness in showing the value of an early eradication of any suspected focus, even when the clinical symptoms are only those of a general septic state, without definite signs of localization, except the very acute persistence of a streptococcic middle ear infection. One is further impressed by the fact that although no classical signs of mastoiditis were present, in each case by exclusion and careful recognition and consideration of every factor, as the meningismus in case 3 and positive blood culture in case 4, our position was made sufficiently strong to urge immediate operation.

XX.

THE NATURE OF NYSTAGMUS AND DIZZINESS OCCURRING IN CERTAIN POSITIONS OF THE HEAD AND IN MOVEMENTS OTHER THAN THOSE AROUND THE LONG AXIS OF THE BODY.*

By Ignatz Sommer, M. D.,
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and
Joseph C. Yaskin, M. D.,
Philadelphia.

A. Report of Cases.
B. Analysis of Clinical Findings.
C. Comment.
D. Summary.

A. Report of Cases.

Cases are occasionally seen where the head is in a certain position and where certain movements of the head and body, although not around the long axis of the body, will produce nystagmus of the peripheral labyrinthine type combined with turning dizziness.

In Professor G. Alexander's Otological Clinic in Vienna, fifty consecutive cases were examined to determine how frequently and in what pathologic condition this syndrome was to be observed.

The fifty cases are divided into five groups as follows:

1. Cases with high degree of nystagmus..... 5
2. Cases with nystagmus in end position..... 5
3. Cases with spontaneous nystagmus increase 2
4. Dizziness without nystagmus..... 7
5. No response to movements..... 31

*Read by Joseph C. Yaskin, M. D., Philadelphia, Pa., by invitation before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, December 19, 1928.

The examination in these cases consisted of the following procedure. During the examinations the patients' eyes were closed to exclude visual images:

(a) Extending head backwards, slowly and rapidly.
(b) Quick movement of head forward and backward.
(c) Stooping forward.
(d) Moving of head from side to side.

SUMMARY OF CLINICAL FINDINGS.

Group I.—Cases with high degree of nystagmus.

Case 1.—P. J., male, aged 18, student. According to the history obtained from the patient, he stated that since five years of age he could, when lying and extending head backwards, observe movements of objects around him. In this position we observed a horizontal rotatory nystagmus of high intensity to the right, lasting about two or three minutes, during which time he had turning dizziness with movement of surrounding objects. This nystagmus could be produced several times every few minutes until the mechanism apparently became exhausted. We succeeded in reproducing it on several occasions and there was no spontaneous nystagmus. A complete examination of ear, eye, central nervous system and all laboratory findings were negative.

Case 2.—Sch. J., male, aged 19, automobile mechanic. For the past year patient observed that when turning his head he experienced turning dizziness with movement of the surrounding objects to the right. There was no spontaneous nystagmus. Movement of the head to the left and the right after a few moments caused horizontal rotatory nystagmus to the left, lasting about a minute. By intensive movements of the head the nystagmus continued on for a longer period. It could be reproduced several times at few minute intervals. The case was observed for four months, and both the nystagmus and the dizziness were reproduced every few days. Complete neurologic, eye, ear and laboratory investigation were negative.

Case 3.—L. J., female, aged 41, houseworker. Patient complained of tinnitus and dizziness which lasted for five weeks. No spontaneous nystagmus was noted. Movement of the head from side to side caused a horizontal nystagmus to the left.

During the nystagmus the left eye would go into convergence. The nystagmus lasted about fifteen seconds, during which time the patient complained of turning dizziness, with a tendency to fall to the right, and showed past pointing to the right. The nystagmus was reproduced three or four times at few minute intervals. This was observed on several different days for a period of weeks. An early lesion of the left cochlear nerve was noted. Both vestibular components were normally irritable. Neurologic, eye and laboratory investigations were entirely negative.

Case 4.—P. E., male, aged 45, locomotive engineer. Patient had symptoms for the past six months. There was no spontaneous nystagmus present. Movement of head from side to side produced horizontal nystagmus of second degree with turning dizziness. The phenomena lasted a few seconds and could be reproduced at several minute intervals. The patient was examined three or four times in the course of a month's observation. He had bilateral occupational cochlear neuritis. The vestibular component was normally irritable. All other investigations were negative.

Case 5.—K. H., male, aged 46, pharmacist. Patient gives a history of having developed indigestion from canned food, after which he noted difficulty in hearing and turning dizziness. He recovered within six weeks but returned later with the same symptoms. At the present time, when lying flat, he experiences turning dizziness which awakens him and the objects seem to turn from left to right. When he extends his head backwards he gets turning dizziness and, in addition, rotatory nystagmus of second degree to the left. The same symptoms occur when he stoops or makes any quick movements. This phenomena lasts about forty seconds; it can be reproduced in few minute intervals. No spontaneous nystagmus present.

Ear examination: Early signs of cochlear lesion on the right side were noted. Vestibular components on both sides were normally irritable. All other examinations were negative, except for evidence of general vasomotor instability. The symptoms disappeared within a few weeks.

GROUP II. CASES WITH NYSTAGMUS IN END POSITION.

Case 1.—H. M., female, aged 40, houseworker. There was no spontaneous nystagmus present. Movement of head forward and backward and from side to side produced horizontal rotatory nystagmus in end position combined with turning dizziness. This nystagmus lasted for a very brief period (five or six jerks) and could be reproduced very often. Patient had a right suppurative otitis media with total destruction of the ear drum. Both vestibular apparatus were normally irritable, and all other examinations were negative.

Case 2.—B. J., male, aged 35, engineer. Fifteen years ago patient had a radical mastoid operation performed on the left ear. At the present time he can hear well, but has a circumscribed labyrinthitis on the left side and cholesteatoma with a positive fistula test. The latter disappeared with the removal of the cholesteatoma as did the previously present spontaneous nystagmus. Movement of the head forward and backward and from side to side caused horizontal rotatory nystagmus in end position, accompanied by turning dizziness. This nystagmus lasted only a few seconds and could be reproduced every few minutes. This was observed at weekly intervals, for two months, when it disappeared entirely. All other investigations were entirely negative.

Case 3.—Sch. K., female, aged 53, houseworker. Patient complained of painful joints. No spontaneous nystagmus was present. Movements of head forward and backward brought forth no response, but movements from side to side caused a horizontal rotatory nystagmus in end position, accompanied by turning dizziness. These phenomena were of very brief duration, could be reproduced every few minutes, and were observed on several different days. Patient showed early signs of bilateral cochlear neuritis, probably of arteriosclerosis bases, as the patient had hypertension and general arteriosclerosis. The vestibular apparatus were normally irritable. Ptosis of the right upper lid and tic of the left face were observed. Wassermann and all other investigations were negative.

Case 4.—S. A., male, aged 48, tailor. For the past few years patient has had left sided chronic otitis media with considerable destruction and granulation. Patient complained of recur-

rent attacks of dizziness. No fistula symptoms were noted. Bilateral cochlear lesion more marked in the left side and vestibular apparatus normally irritable on both sides. No spontaneous nystagmus observed. In all our movement tests the patient got nystagmus of first degree to both sides, accompanied by turning dizziness. This lasted for about 30 seconds and could be reproduced every few minutes. All other investigations were negative.

Case 5.—W. F., female, aged 28, housemaid. Patient sustained a blow on the left ear a few weeks prior to examination and soon after complained of tinnitus and difficulty in hearing. No spontaneous nystagmus observed. Evidences of left cochlear involvement; both vestibular apparatus normally irritable. In all our test movements patient revealed horizontal nystagmus to the left of first degree, accompanied by falling to the right and turning dizziness. This phenomenon lasted only a few seconds, reproduceable every few minutes, and persisted for a period of several months. All other investigations were negative.

GROUP III. SPONTANEOUS NYSTAGMUS INCREASED.

Case 1.—L. A., female, aged 32, saleslady. Patient had a right-sided chronic otitis media and left-sided dry perforation of several years' duration. She had spontaneous nystagmus to the right. When looking to the right she experienced about six or seven jerks, and movements of the head forward and backward increased the jerks to about fifteen. There was an accompanying turning dizziness. This could be reproduced at few minute intervals and was observed on several different days. Movements of the head from side to side did not change the spontaneous nystagmus. The cochlear and vestibular tests were negative. Apart from healed tuberculosis of the left knee all the other tests were entirely negative.

Case 2.—S. A., female, aged 28, factory hand. During childhood patient had otorrhea of right ear which remained dry until one year ago, when a fibroma was removed from right soft palate and the ear again began to discharge. In the past few days she had had turning dizziness with pain in right ear. A right-sided suppuration of the antrum with spontaneous perforation in the posterior part was noted.

Right ear whispered voice, six meters—cochlear test middle ear lesion; left ear whispered voice normal—cochlear test normal. Spontaneous nystagmus, horizontally rotatory to left first degree—falling to right, past pointing to right. There was tenderness over right mastoid. Caloric tests revealed normal vestibular functions.

Movements of the head, anterior and posterior and from side to side, resulted in horizontal rotatory nystagmus to both sides, which was of considerable duration and was accompanied by turning dizziness. Pressure on tragus on right side caused dizziness and nystagmus to both sides.

Operative findings: Conservative radical operation. The antrum and attic were widely opened and covered by a flap from the external auditory meatus. Findings: The lateral wall of the labyrinth appeared to be normal, smooth bone. Three weeks after the operation patient still had spontaneous nystagmus. Forward and backward movements of head still increased nystagmus and produced dizziness.

GROUP IV. DIZZINESS WITHOUT NYSTAGMUS.

Case 1.—D. J., male, aged 54, laborer. Patient complained of falling to right, which was diagnosed as coronary sclerosis with generalized arteriosclerosis. On our test movements he had turning dizziness but no nystagmus. Evidence of cochlear hypofunction of right side and marked dizziness upon Kobrak's caloric test. All other investigations were negative.

Case 2.—T. J., male, aged 28, clerk. Patient complained of attacks of turning dizziness which were of brief duration. Examination of the optic papilla left some doubt as to whether the condition was a pseudoneuritis or early choked disc. Neurologic examination was entirely negative. Examination of the cochlear and the vestibular components were likewise negative. Movements of the head forward and backward were negative, but movements from side to side caused turning dizziness. All other investigations were negative.

Case 3.—O. M., female, aged 35, houseworker. Patient complains of Ménière-like attacks, lasting three to four days, occurring at irregular intervals for a period of several years.

When examined during the attack there was evidence of considerable cochlear hypofunction, with failure of vestibular response on both sides. After attacks there was a return of both hearing and vestibular irritability. In the intervals between the attacks all our test movements produced turning dizziness but no nystagmus. The patient smokes to excess. A most careful survey by clinical and laboratory methods was entirely negative.

Case 4.—W. L., male, aged 56, leadworker. Patient had a chronic right-sided otitis media of 30 years' duration. Second position nystagmus was of very brief duration, and there were present five to six jerks. There was a bilateral cochlear involvement but normal vestibular irritability. Our test movements produced no change in the nystagmus, but were followed by considerabel dizziness. All other investigations were negative.

Case 5.—R. A., female, aged 50, houseworker. For the past few weeks patient complained of attacks of turning dizziness and ringing in the ears. Examination revealed horizontal rotatory nystagmus in end position of moderate duration. There was a bilateral cochlear lesion and a hyperirritable labyrinth on both sides. Movements of head forward and backward were without effect. Movements of head from side to side produced pronounced turning dizziness, but no change in the nystagmus was noted. Apart from very lively knee jerks all laboratory and clinical investigations were negative.

Case 6.—St. M., female, aged 31, factory hand. Patient has a bilateral chronic otitis media with ringing in the ears. Movements of the head forward and backward produced no dizziness. Movements from side to side and stooping caused pronounced dizziness but no nystagmus present. Cochlear and vestibular component intact. All other objective examinations were negative and the case was considered as one of neurosis.

Case 7.—Sch. F., female, aged 60, dressmaker. Patient has a left-sided dry perforation of the ear. All test movements showed considerable turning dizziness but no nystagmus noted. Cochlear and vestibular component intact. All other investigations were negative.

GROUP V. NO RESPONSE TO TEST MOVEMENTS.

Cochlear lesion secondary to middle ear	3
Cochlear lesion due to arteriosclerosis	8
Cochlear lesion, hereditary	1
Cochlear lesion, postinfluenzal	1
Cochlear lesion, toxic	2
Cochlear lesion, postencephalitic	1
Cochlear lesion, occupational	3
Cochlear lesion, luetic	2
Cochlear lesion, traumatic	1
Cochlear lesion, syringomelia	1
Cochlear lesion, climateric	2
Cochlear lesion, other causes	3
Chronic otitis without cochlear lesion	3
	31

Of thirty-one cases three had a hypoirritable or lost labyrinth function while one had a hyperirritable labyrinth.

B. Analysis of Clinical Findings.

An analysis of our cases is fruitless in so far as our ability to establish any definite etiologic relationship between nystagmus and dizziness on the one hand and some definite clinicopathologic condition on the other is concerned.

The nineteen cases in groups I to IV, in which combined nystagmus and dizziness or only dizziness occurred, were essentially normal so far as the eyes, the nervous system and general somatic conditions were concerned, as investigated by the usual clinical and laboratory methods. As far as the ear was concerned, all except one of the nineteen cases had a normally irritable labyrinth. As to the cochlear component of the nineteen cases, ten showed unilateral or bilateral cochlear hypofunction, which is not very significant, when one considers that of the thirty-one cases in group V (in which there was neither nystagmus nor vertigo) twenty-eight had a definite cochlear hypofunction. Of the nineteen cases in groups I to IV, inclusive, only seven had chronic middle ear processes, while in group V there were six cases with middle ear disease—perhaps a preponderance of middle ear conditions in our positive cases.

Of the nineteen cases in groups I to IV, twelve cases had combined dizziness and nystagmus, seven only dizziness. Of the first twelve cases named, seven had a cochlear hypofunction and eight had chronic middle ear disease. Of the cases with only dizziness, three had middle ear disease and two definite cochlear hypofunction, while two other cases had Ménière-like attacks.

C. Comment on Mechanism of Production of Nystagmus and Vertigo in These Cases.

I. Nystagmus.—The involuntary rhythmic oscillations of the eyeballs, the nystagmic movements, are in reality defensive reactions of the ocular muscles in the interest of optical fixation and orientation in space. Normal function of these muscles—that is, the ability to fixate on a given object depends upon calculated correct movements and upon the ability, after bringing the object in line of the fovea centralis of both eyes, to keep them there for the necessary period, which is accomplished by normal tonus. The production of these movements and the necessary tonus depends, on the one hand, upon voluntary innervation, and on the other, upon certain reflex phenomena, the afferent arm of which is initiated principally in the retina and in the labyrinth (Stellungapparat and Blickapparat—Cords[1]). Any factors, clinicopathologically or experimentally induced, by interference with visual fixation or labyrinthian function, may produce nystagmus. Everyone is familiar with the experimental optical nystagmus (Bárány[2]—Brunner[3]) and the many varieties of ocular nystagmus as seen in the blind, in the amblyopic, in certain hereditary cases, in miners' nystagmus, etc., and in the closely related Endstellungnystagmus (Cords[1]), etc., all of which have no involvement of the peripheral or of the central labyrinth.

In this presentation, however, we are more interested in the other component concerned with eye muscles in their relation to tonus, position and movements. In so far as the tonus and position of the eyes and the necessary reflex movements for maintaining position (Stellerreflexes) is concerned, it is fairly well established that in most animals (Magnus[4]) and in all probability in man (M. H. Fischer[5]), these functions are closely related to the otolithic apparatus and, therefore, on the position

of the head, in which the neck muscle reflexes play a significant role.* The semicircular canals appear to exert no influence on these more or less static functions (Magnus[4]). On the other hand, in the matter of coordinated eye movements the semicircular canals with the various central connections are considered of prime importance (Ewald[6]). Indeed, it is the disturbance in these pathways that gives rise to the most important types of nystagmus, the peripheral labyrinthine and central types of nystagmus. The part played by the otolithic apparatus is not definitely known, either on the experimental or clinical side. What is generally assumed is that movements in the endolymph produced by caloric irrigation, turning and by compression or decompression of air (fistula test) induce nystagmus which takes its origin in the semicircular canals. Further proof of this rests on the experience that the type of nystagmus changes with position of the head, which determines the choice of the particular canal stimulated by the experiment. The dependence of the action of a given semicircular canal upon the position of the head may be of interest in the consideration of our problem.

So that we are justified in saying, in the present status of our knowledge, that in so far as the peripheral labyrinth is concerned, apart from the vestibular nerve itself, the semicircular canals are of first importance in the production of nystagmus. Of course, it need only be mentioned that the central nystagmus may have its origin from lesions in the nuclei of the brain stem and its connections with the posterior longitudinal bundle as well as in the latter itself.

In normal individuals it requires movements of a certain intensity and around the long axis of the body to produce nystagmus—that is, to produce sufficient current in endolymph. Likewise ordinary movements of head with eyes shut (to rule out retinal influences) are not capable of producing nystagmus, though Bárány believes that these movements always cause nystagmus which disappears immediately upon cessation of movement. Though the last named author's observations have not been confirmed, other evidence goes to prove that semi-

*The above statement does not, of course, include such simple disturbances as loss of voluntary innervation and other conditions which may interfere with maintenance of normal position.

circular canals are rather sensitive under certain circumstances. Thus only slight localized labyrinthitis, paralabyrinthitis or a mere adhesive process (Brunner[8]), in the middle ear can give rise to nystagmus, although usually this is inconstant, transitory and, as may be expected in an irritating lesion, its direction is toward the affected side. Just why the semicircular canals are so sensitive is hard to determine. It is not unlikely that the cause is to be found in the blood supply of the inner ear. Blood vessels of the middle ear are continuous with blood vessels of the inner ear through the bony partition (Politzer[9]), and the involvement of the middle ear may extend to the inner ear. Kobrak[10] believes that vasomotor change in the inner ear may produce labyrinthine symptoms, both through pathologic changes and clinical experimentations. However, it may be, it is conceivable that whereas the normal semicircular canals are resistant to movements such as we used in our tests, the sensitized or susceptible inner ear permits a more ready disturbance of endolymph circulation, and by thus lowering the threshold permits ordinary movements or change of position of the head to cause nystagmus.

The localization of the origin of nystagmus in the semicircular canals is not undisputed. Thus Voss[11] described nystagmus and vertigo in certain head positions and ascribed it to disturbances in the otolithic apparatus. By fixation of the neck in plaster of paris, he ruled out the neck muscles and established the otolithic apparatus by "gegenrollung" of the eyes in certain positions of the head. The case is one of acute labyrinthitis in which special localization could be of very little value, because of the involvement of both semicircular canals as well as of the otoliths. Stein and Brunner[12] think that this type of nystagmus is caused by action of the neck muscles acting upon some hypothetical lesion in the brain stem by way of the spinocerebellar tracts and cerebellum. Unfortunately the authors gave no basis for their localization of the foci in the brain stem and no reason for assigning the spinocerebellar pathway as agent in production (cerebellovestibular pathways; inhibitory!). Voss[11] ruled out the neck muscles as pointed out above.

Baldenweck[13] comes to the conclusion that the slow head movements produce nystagmus by irritation of the otolithic ap-

paratus, while rapid movements produce it by action on semicircular canals. There is no definite clinicopathologic evidence in favor of the former view, while experimental evidence is decidedly against it.

Some of the earliest observers, notably Bruns[14] and Oppenheim,[15] ascribed nystagmus in certain positions and movements of the head as being pathologic and usually signifying cysticercus of the fourth ventricle or cerebellar tumors. The same view was held by Bárány,[7] who added to the two conditions mentioned multiple and circumscribed labyrinthitis. Marburg[16] is of the same opinion, based on an autopsy finding. It is readily conceivable that a tumor may cause mechanical pressure or sufficient circulatory disturbance by movements or change of position, to produce nystagmus by either irritation of the central pathways or possibly the nerve stem. While the possibility of disease of the central nervous system should always be borne in mind when one sees cases of this type, we believe that in our group we were dealing with the peripheral labyrinth and more particularly with the semicircular canals.

II. Dizziness.—Anyone who has ever attempted to investigate the subject of dizziness will realize its many difficulties. Most authors seem to agree that it is a psychic process associated usually with an unpleasant feeling tone (rarely pleasant), a feeling of movement perceptions or perhaps deceptions in relation to oneself or the surrounding world, and accompanied by such disturbances of vegetative nervous system as sweating, vomiting, etc. Viewed from a psychophysical standpoint it is a form of disturbance of balance. This depends upon three important factors: (a) optical perceptions, (b) proprioceptive sensibility (including skin, muscles, joints, etc.), and (c) labyrinth. According to R. Leidler,[17] the labyrinth is the "final common pathway" for the other efferent arms—that is, the optic and proprioceptive. In addition, this author regards the cerebellum as the inhibitory organ of the labyrinth, while E. Spiegel[18] connects the vestibular apparatus with the cortex by way of the red nucleus and thalamus. Accordingly, a disturbance in any of these connections is apt to produce vertigo, and it is more than likely that the particular arm affected causes a special type of dizziness. Thus in retinal disturbances there is a sensation of movements that have no particular direction,

while in disturbance of the proprioceptive component there is a sensation as if the "Unterlage" has disappeared, abnormal weight of the head, etc. The above types of dizziness are generally indefinite and variable. On the other hand, the types associated with lesions in labyrinthine pathways (central and peripheral) and of the cerebellum are of "Tastschwindel" or both. Dizziness of this type may also occur in central disease, but its exact mechanism is not clearly understood. The practical point to remember is that "turning" dizziness, with or without "Tastschwindel," means the involvement of (a) the peripheral labyrinth or vestibular nerve, (b) the nuclear masses in the brain stem and their central connections, or (c) the cerebellum.

Now in our nineteen cases the dizziness was of distinctly this type (turning). There was nothing in our finding to point to involvement of the cerebellum or of the central vestibular pathways, and we are inclined to the opinion that in our cases the vertigo, like the nystagmus, is of peripheral labyrinthine origin. We have the feeling that increased susceptibility to minimal stimuli, which causes the transient nystagmus, in all probability causes the labyrinthine vertigo. We have no knowledge of any localization of the process in the labyrinth.

D. Summary.

1. Fifty consecutive cases were investigated in an otologic clinic in Vienna to determine the occurrence of nystagmus and vertigo in certain positions of the head and in head movements other than those around the long axis of the body. Of these, twelve cases had combined nystagmus and vertigo and seven only vertigo; thirty-one gave no response.

2. No etiologic relationship could be established between these phenomena and any clinicopathologic condition in our patients. Of the nineteen cases, all except one had normally irritable labyrinths. Ten cases had cochlear hypofunction and seven chronic middle ear processes forming no striking contrast with the thirty-one cases which did not reveal either nystagmus or vertigo.

3. Our investigations would assign the immediate cause of these phenomena to increased susceptibility of the semicircular

canal component of the labyrinth. This does not mitigate against the occurrence of these signs in lesions of the brain stem and the parts of the central nervous system, as reported in literature. In our cases, however, there was no evidence of any such lesions.

4. These two signs by themselves are not indicative of a serious condition either in the peripheral ear or in the brain stem. In themselves they are not sufficient indications for establishing diagnosis of a serious condition either in ear or brain stem and are not indications for surgical interference.

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XXI.

A POSITIVE SYMPTOM OF MASTOIDITIS.

By Murray Berger, M. D.,

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A positive symptom of mastoiditis which appears frequently is spasm and rigidity of the upper third of the sternocleidomastoidius muscle. This symptom is most marked and is found more often in children. It is seen more often in those adults whose mastoid cortex is thin or whose mastoids have been perforated. The spasm and rigidity depend upon the extension of the suppurating process. In an acute suppurative otitis media there is mastoid pain and tenderness, but never spasm and rigidity, as the only pathology found is red and swollen mucous membrane lining the cells. Spasm and rigidity are found only when the mastoid cells have undergone necrosis.

This symptom is best elicited by gently palpating with the fingers of both hands and comparing both sides. It is only of significance when it is present, and the absence of this symptom means nothing, so that the diagnosis of the underlying conditions will have to be made on the associated symptoms.

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XXII.

CHRONIC LARYNGEAL STENOSIS COMPLICATING LARYNGEAL DIPHTHERIA.*

By R. J. Payne, M. D.,

St. Louis.

Direct laryngeal observation by the use of the electrically lighted laryngoscope has greatly facilitated the study of diseases of the larynx, especially in small children, where the use of the laryngeal mirror is most difficult and in many cases practically impossible. With a child rolled in a mummy sheet, head projecting from the table, and held in proper position by an assistant, the laryngoscope can be introduced into the larynx with little or no trauma and with comparatively little discomfort to the child. We have not found it necessary to administer general or local anesthesia to facilitate either examination or instrumentation.

The general condition of a child suffering from the effects of laryngeal diphtheria would in itself militate against any sort of manipulation which would tend to produce increased heart strain. In cases in extremis where there is great air hunger, any examination other than to give instant relief and make a diagnosis, would not be undertaken. In the hands of one skilled in the introduction of the direct intubation tube through the laryngoscope, there is opportunity to observe local conditions not afforded by other methods.

Dr. Lohr and I have had opportunity to study by this method, new to this institution, 77 cases of laryngeal diphtheria. We have found it of particular value in the observation and treatment of complications, particularly chronic stenosis.

Ulcerations of the larynx from the presence of the intubation tube may arise very early. Pressure necrosis and injury to the deeper structures are sure to follow the continued use

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of the intubation tube beyond the allotted time. The result is stricture.

Intubation tubes are of arbitrary sizes. They correspond to the size of the larynx, according to the age of the child, but there is a great variation in size of the larynx of a large or small child, even of the same age. No method has been devised to ascertain the amount of edema or membrane present. Within the last few years we have received three cases from the same physician; one child wearing an intubation tube for over three weeks, the other two for over four weeks. Tracheotomy was at once performed with removal of the intubation tube. In each case the direct laryngoscopic examination showed the presence of pressure necrosis. We prefer tracheotomy to the continued wearing of an intubation tube in all cases requiring reintubation for more than six or seven times, as subsequent intubations greatly enhance the possibility of chronic laryngeal stenosis.

Chronic laryngeal stenosis has occurred in cases with acute stenosis admitted under the diagnosis of laryngeal diphtheria. These cases failed to show positive culture, even on repeated examinations. This leads us to believe that there is an underlying mixed infection responsible for the stenosis, even in laryngeal diphtheria. This, however, has not been definitely proven.

The following types of cases suggest a diagnosis of chronic stenosis:

1. Patients wearing an intubation tube for over two weeks. We have found tracheotomy much safer for the patient. It allows the larynx to recover from the trauma and often prevents stricture.

2. Patients requiring repeated intubations at decreasing intervals of time.

3. Patients already wearing tracheotomy tubes who develop air hunger on plugging of the tube.

4. Patients who gradually develop difficulty in breathing after having worn an intubation tube months previously and having apparently recovered. Examination of these cases shows narrowing of the laryngeal aperture.

In laryngeal stenosis there is a characteristic hoarseness and brassy voice together with difficult breathing. In more severe

cases the accessory respiratory muscles are brought into play, coupled with suprasternal depression. The latter is regarded of great significance.

LARYNGEAL PICTURE.

1. The superglottic type shows an edematous or warty appearance over arytenoids and aryepiglottic fold. The redundant tissue obstructs the view into the larynx but produces little resistance to instrumentation. It is easily pressed aside. There is no gripping of the dilator as in the case of cicatricial stenosis. On withdrawal of the sound this exuberant tissue falls into the lumen of the larynx and again obstructs breathing.

2. The subglottic type occurs below the level of the vocal cords, in an area rich in blood supply, which is no doubt a factor in the prevalence of stenosis.

3. The type involving the more central portion of the larynx is accompanied by extreme hoarseness or aphonia.

All stages of inflammation are exemplified in these conditions. The inflammatory process, if it has advanced, extends through the mucosa into the cartilaginous framework. The perichondritis and thickening obliterates the lumen of the larynx and perhaps produces the worst form of obstruction. Next, the loss of tissue by ulceration caused by pressure necrosis from the intubation tube results in fibrous tissue replacement, producing an organized stricture. Webbing or thin adhesions are often seen over the lumen. These are easily penetrated and ironed out by use of the laryngeal sounds.

1. Tracheotomy, in lieu of intubation, prevents pressure necrosis, and in mild types leads to early recovery.

2. In severe cases, where the edema fails to subside, superficial cautery has been employed with fair results.

3. Cicatricial strictures have been ironed out by the use of Jackson's triangular laryngeal dilators, sizes 14 to 36, successively employed. This procedure requires cooperation between the operator and assistants. As the sound is passed through the laryngoscope and into the larynx, under direct view, the tracheotomy tube is partially withdrawn and is immediately replaced upon removal of the sound. It is, of course, impossible to dilate the larynx with tracheotomy tube in situ.

We have failed to relieve the stenosis in a few cases. They have resisted all forms of treatment. Probably laryngofissure and skin grafting is the only alternative. But the general consensus of opinion is that a conservative course should be followed.

If the patient can breathe without difficulty when the finger is placed over the lumen of the tracheotomy tube, the tube is corked. We first use a plug occluding one-half the tube. If this can be comfortably worn for 24 hours, a second cork is inserted, occluding three-fourths of the tube. When the tube can be comfortably worn for 24 to 36 hours with lumen entirely plugged decanulation may be safely undertaken.

718 University Club Bldg.

XXIII.

TOXIC OTITIS.

By J. J. Hompes, M. D.,

Lincoln, Neb.

Most otologists have at least some knowledge of the voluminous literature which exists relative to ear conditions that result from constitutional disorders, but it is my belief that many of us make little application of that knowledge in the routine handling of cases.

How many men, after making a diagnosis of eighth nerve deafness, will explain to the patient the necessity of a general physical examination? I am sure that there are many practitioners to whom such an idea almost never occurs, and the same criticism could be made of most of us, at least a part of the time.

It is the observation of those making careful and complete studies of such cases that far too many men give their entire attention to the removal of the primary foci of infection to be found in the field of their specialty, and even though infection cannot be demonstrated some operative interference is made, either in the nose or throat, with a hope that some good will be done. In the cases where positive infection exists and is removed beneficial results often do not follow, and the superficial doctor and the patient soon become discouraged and quit when just a little more thinking might lead to the discovery of a possibility for a satisfactory result.

It is a strange thing that most nose and throat operators will admit that they can often do something for a rheumatic knee or a cardiac disease by taking away an infected tonsil and thereby removing its infectious and toxic elements from the blood stream, and yet how many of those same operators seem to think that when they take an infected tonsil from the neighborhood of an infected ear that the result is accomplished in an entirely different way? Their action in the face of failure would lead one to believe that the ear was the sufferer through

continuity of tissue rather than through the heart's blood, as in the case of the knee or other more distant part. I believe that such a conclusion is justified, otherwise they would not leave out of consideration the fact that the primary foci which they have removed might have affected some other organ or tissue and that possibly this other organ so affected might be causing the ear symptoms. Few doctors would expect every case of thyroid gland or gall tract disease to clear up just because the exciting factor, say an infected tonsil, had been removed. A lack of visualization of the modus operandi for results where results are obtained by simple removal of the primary foci begets unforgivable thoughtlessness when such a procedure ends in failure.

If you were to ask most physicians whether or not a toxic eighth nerve neuritis could come from a Graves disease or a cholecystitis, his answer would be "Yes," and he might recall that Wittmaack, in his article on the subject in 1904, drew the following conclusions, namely: "No longer can any doubt exist as to the existence of an acoustic neuritis analogous to an optic neuritis or a toxic polyneuritis. The etiology on which acoustic neuritis is based is the same and as multiform as the etiology of the two other diseases," and may I add at this juncture that many of the otalges and acute and chronic catarrhal otitis media cases have the same multiform array of factors in their etiology as does acoustic neuritis. I believe that most any physician would say that he believed that ear symptoms are possible in any general organic disturbance, and in the same breath he could honestly admit that he has made little use of the fact.

I suppose that the answer for our many shortcomings in this way lies in the fact that in our special training teachers have stressed methods for functional testing as a means of diagnosis and have taken it for granted that we would apply our general knowledge of medicine in addition to those methods. A compliment to quite an extent undeserved. At any rate, it is perfectly obvious that a careful and complete study is as necessary in otologic cases as is ophthalmologic, urologic or other cases of organic disease. Of course, the economic factor enters in. All cases cannot stand the expense of a complete physical examination nor is such an examination necessary in every case. The otologist, if he will take careful histories, gen-

eral as well as special, get the temperature, the quality and rate of the pulse and the blood pressure, will be able to pick out the cases in most instances that require a detailed study. If such a simple general examination is done in every case, it is astounding how many cases of serious disease the otologist will get diagnosed in their incipiency. I have picked up a great many unsuspected cases of pulmonary tuberculosis and thyroid disease that came in with ear symptoms, just on the pulse, temperature and blood pressure findings, together with the history elicited either before or because of these findings. Patients are easily referred for physical examinations if you have established a definite basis for your advice.

In taking temperatures it is well to remember that low grade temperatures do not register in two minutes. The thermometer should be left for at least eight minutes. If the otologist is competent to make more complete examinations I see no reason why he should not do all for his patient that he is capable of doing.

The following case reports are given to emphasize the foregoing statements. Similar ear cases from the same causes are to be found in the literature by otologists from everywhere with many other types of cases of which I have had no examples and of which I shall make no mention. It just so happens that in most of the contributions only one case is reported or the study of one disease or a toxin make up the report. It may be that it is for this reason that some get the idea that such cases are unusual and therefore fail to give the reports the consideration they deserve. I am for this reason including a number of cases from different causes.

Case 1.—Mrs. A. W. K., age 28. Hard of hearing before having influenza, three years ago, since which time she has had tinnitus in both ears. She has pain a great deal of the time over left maxillary region and left mastoid.

Bone and air conduction both shortened. Tonsils removed and nose and sinuses were negative. Pulse 140, temperature 100° F., blood pressure 100/68.

After these findings the patient admitted that she had lost some weight and that she was tender in the region of her appendix. She was referred to an internist, who made a diagnosis of chronic appendicitis, and the appendix was removed three

or four days later. I have not seen the patient since, but a letter received from her within two months of the time of her operation assured me that she would not have to return on account of her ears, as her head noises were gone and her hearing had returned.

It is hard to tell in this case how much of her ear trouble was due to toxic neuritis and how much was due to a low blood pressure coming from a toxic myocarditis. The short recovery period and the extent of recovery would make one think that the symptoms were due to a physical rather than a pathologic condition of the ear. One other thing in this case to which I wish to call your attention was the presence of pain over a sinus and the mastoid process. Pain, boring pain, through the mastoid processes and in the region of the sinuses, is common in toxic conditions.

Case 2.—Miss E. R., age 22. Progressive loss of hearing for five years, much worse recently. Could hear conversational voice at eight feet in either ear. Diagnosis of nerve deafness was made. Nose and throat negative. Tonsils had been removed. Her temperature was 100° F.; blood pressure 135.

Referred for physical examination, and a bilateral pyelitis was found. No other physical defect being uncovered at the time or in several subsequent examinations by several competent men. Two urologists have tried to cure her pyelitis without result and have told me that the case was not remedial so far as they were concerned.

I have seen this case many times during the past five years. Her hearing has gone down until she hears a loud voice at contact only, and she always has a temperature near the hundred mark, a headache and high blood pressure.

Case 3.—Mrs. H. L., age 45. Complained of fullness in ears the last six or eight months. Has not been well for several months. Had appendix out a few months ago. Has felt worse since. Had some infected teeth out shortly after appendix operation because she did not get strong after the appendectomy. She said that the ear trouble began when her health began to fail.

There were no other ear findings than shortened bone conduction. Pulse 96, temperature, p. m., 99.2. Final diagnosis, pulmonary T. B. I do not know the final outcome but do take

the credit for getting a proper diagnosis in a much abused case.

Case 4.—Mrs. R. B. S., age 50. Came in complaining of some difficulty in hearing during the past few days. She said that she had been having head noises for some time. She said that her family physician wanted to take her tonsils out to cure her ear condition.

A diagnosis of catarrhal eustachian salpingitis was made. All of her pharyngeal mucous membranes were red and boggy, with a great amount of abnormally thick secretion. The typical picture of a mucous membrane under general toxic conditions.

The woman was over weight. Her blood pressure low, 110; pulse 78; temperature high, 99.3. Final diagnosis, cholecystitis with stone, which was operated upon with relief of all symptoms. She still has the tonsils, so far as I know.

Case 5.—Mrs. A. J. B., age 39. Complaint, ringing in both ears for past two weeks. Has had tonsillitis frequently, has rheumatism at times.

Findings: Deflected nasal septum, infected tonsils, infected teeth, no ear findings. Temperature 99.4, pulse 120, blood pressure 180. The final diagnosis was Graves disease, for which proper treatment was accepted with good results. This woman admitted that she had been pretty nervous for some time but thought the nervousness due to hard work.

Case 6.—E. S., age 16. Brought in by parents, who stated that the child's hearing had been defective for several months. He had the same trouble three years previously, which had lasted two or three months. His hearing for conversational voice was five feet in either ear. Bone conduction shortened on both sides.

He had infected tonsils and adenoids. Temperature 98.8, pulse 86, blood pressure 140 and W. B. C. of 16,000. Albuminuria and pyelitis.

Removal of the foci of infection and treatment of the pyelitis effected a cure in a few weeks.

Case 7.—C. C. M., age 38. Had defective hearing in left ear since early childhood, thought to be brought on by tonsillitis which he had frequently as a child.

His present trouble came on following influenza in 1918. He complained of deafness, dizziness and vertigo with inability to walk without staggering and inability to retain food or

water for a day or two at a time. The attacks came on two or three times a month. He complained of severe head noises all of the time and stated that they became almost unbearable during the attacks of dizziness and vertigo. He had seen several physicians, and one otologist consulted is one of the outstanding men in the country. He received no relief and went to Florida for the winter of 1919-20, where he was comparatively comfortable and his hearing improved. He came to his farm home in Nebraska the last of March, 1920, and became worse again. He had had infected teeth and tonsils removed previously without result, and physical examinations, including spinal puncture, and complete laboratory examinations revealed no cause for his malady. He always had a subnormal temperature and a blood pressure around a hundred. I put him on adrenalin chlorid treatment, recommended by Dr. Joseph Beck, which made him worse. I finally induced him to try another internist, Dr. John C. Thompson of Lincoln, who determined that he had incipient T. B. involving both apices. The patient went to bed for two months on a T. B. regime, with the result that he picked up seventeen and one-half pounds in weight, lost his Ménière's syndrome and made a remarkable gain in hearing. The Ménière's symptoms returned for a short time two years later when he was overworking. The gain in hearing was only temporary, and a progressive loss in hearing resulted until he is now only able to hear a loud voice at contact. One has a right to believe that hearing might have been saved for this man if the diagnosis could have been established in the beginning.

Case 8.—Miss K. M., age 22. Another case with a Ménière's syndrome for one year. She had been told by a well-known otologist, that she had Ménière's disease and given a bad prognosis. The internist to whom I referred her reported chronic appendicitis with W. B. C. of 15,000. The ear symptoms and the appendix troubles cleared up together under the dietary and hygienic treatment instituted. Operation was refused.

This case reported three years later with pain in the right ear which was found to be caused by an impacted third molar. She has had no return of her former ear trouble.

Case 9.—Mrs. E. G. L., age 37. Tinnitus right ear and progressive loss of hearing over a period of several months. Had

lost some weight during this period. One sister in a Colorado Sanitarium suffering from pulmonary T. B. Her temperature was 99, pulse 116. Blood pressure was not taken. Ear diagnosis, eighth nerve affection, based on shortened bone conduction. Internist report, active pulmonary tuberculosis. This patient spent six months in bed with excellent results. I have seen her frequently in the six years that have elapsed, and she says that she has had no ear symptoms since the first few weeks of her rest period for her chest condition.

Many other cases of eighth nerve affection have been seen in patients suffering from pulmonary tuberculosis, for they are common. Bezold in his text of Otology (page 281), says: "Polyneuritis acustica of consumptives is the best known of all diseases of the auditory nerve due to infectious diseases."

I recall one girl who came to me complaining of progressive deafness who became highly incensed when, in taking her history, I inquired as to whether or not there were other cases of deafness in her family. Her answer was "yes," and on both sides of the family at that, and she supposed I would tell her what every other doctor had told her about hereditary ear troubles. When the history was completed I found out that there had been an astounding incidence of tuberculosis in both families, and she proved to be suffering from the malady at the time, with a temperature of 100 F. and a shortened bone conduction, with no other ear findings.

Case 10.—Mr. T. R. L., age 50. This man had a severe Ménière's syndrome with nystagmus, past pointing and falling to the right. He was a chronic alcoholic and a heavy user of tobacco. He had an infected right maxillary sinus, some infected teeth and cholelithiasis. He was relieved of most of his trouble by removal of foci of infection and by leading a more hygienic life. He had a mild return of symptoms occasionally due to his gall bladder trouble, from which he died about a year after his first head symptoms.

Case 11.—Mrs. G. L., age 37. Gave a history of a sensation of fluttering of her ear drums for the past year. She had seen several otologists who had used inflation and massage without result. She said that the constant fluttering was making her so nervous that she could hardly think.

There were no ear findings and I made the guess that she was having clonic spasm of her intrinsic ear muscles. Her temperature was 99, pulse 94, blood pressure 100. Her general appearance was that of robust health. The internists' findings of importance were metabolism minus forty, loss of both ovaries by operation, valvular heart disease and purulent endometritis. The internist has been able to do something for this woman's ear condition.

I have seen a number of cases of toxic neuritis from other causes, but I believe that a consideration of the value of inflation and massage in this last case is sufficient evidence on which to conclude that we are good otologists only when we are good physicians.

XXIV.

UNILATERAL BLINDNESS: RECOVERY AFTER SINUS OPERATION.

By J. T. Dowling, M. D.,

Seattle.

My subject is an old one and much has been written on it. The elder Ballinger was one of the first in this country to call attention to the possible relation between sinus disease and eye complications. He was unjustly ridiculed by many. After him came such eminent American rhinologists as Mosher, Skillern, Dean, Sluder, Sewall and others, who have preached and insisted on the relation of nasal focal infection to eye disease.

On October 2, 1928, Mrs. J. W. T. (Case No. A-28752), age 26, married, was referred to me by her family physician. Her chief complaint was of rather gradually failing sight in her left eye without other subjective symptoms.

Examination: The patient was a small woman, weighing 110 pounds. She was the mother of two children, both of whom are living and well. In 1923 she underwent an hysterectomy and appendectomy with good recovery, and since then has had no pelvic disturbances. A complete physical examination by an internist elicited the fact that she fainted easily: that she had stomach sickness at times, was very nervous and unable to do her work as easily as formerly.

No gastrointestinal study was made, as it did not seem indicated. Her appetite was poor. She was not troubled with constipation. Urine examination was reported negative, as was the Wasserman reaction on the blood. Her blood showed a white count of 9900 per cm. Hemoglobin was 75 per cent. All reflexes were normal. Her blood pressure was systolic, 120 mm. Hg.; diastolic, 70.

Special examination: The eyes were normal in appearance and movement. No pain was referred to the eyes or to the face. She had headaches. One year ago, following extraction of a tooth, she saw some "black spots" before the left eye for a time. These spots disappeared eventually. September 1, 1928,

she noticed that the vision of the left eye was dimming gradually, until on October 2nd, when I first saw her, there was in that eye light perception only. The vision of the right eye was 6/6, of the left eye light perception only. Ocular tension was normal and there was no tenderness of the eyeballs to touch. The pupils reacted to light. The retinal light reflex was present in the right eye but not in the left. On examination of the eyes with the ophthalmoscope, the right retina was normal, but we were unable to see the fundus or any part of the retina of the left eye on transillumination. The right antrum was dark, the left antrum clear, as were the frontal sinuses. A roentgenogram of the sinuses was made and showed both antra dark. The tonsils had been removed. The ears were reported normal in function and appearance. A roentgenogram of the mastoid regions was reported negative. The teeth were reported as having slight pyorrhea of the lower incisors but no apical abscesses.

A tentative diagnosis of the vitreous opacity due to retinal hemorrhage was made, with empyema of the maxillary sinuses as the probable cause. Radical operation on both antra was advised.

On November 4, 1928, under local anesthesia a radical operation on the right maxillary sinus was performed. Some pus and much inflamed sinus membrane was removed with the curette and a permanent opening for drainage into the nose was made beneath the lower turbinate bone. The wound in the mouth was sutured without drainage, drainage having been established through the nose. The antrum was packed lightly with one-half inch strip gauze. The patient made a good recovery from this first operation, and on November 11th the left antrum was operated on with much the same findings. The same procedure was followed as in the earlier operation, but anesthesia was induced by the administration of nitrous oxid, oxygen and ether. As before, the recovery was uneventful and in ten days the patient stated that the vision in her blind eye was improving, although I was unable to prove this objectively. She has been observed once in every ten days ever since the latest operation, and the improvement of her general health and of her vision has been marked. Within two weeks after her second operation light reflex could be obtained in her blind

eye, and she could see large objects, as the hand, at five feet. Now a rather hazy view of parts of the retina can be had but not much detail can be made out. I hope to report, at some future time, the favorable progress of this patient and complete return of useful vision.

In going over the literature concerning focal infection and its relation to the eye and vision, one is astounded at the mass of material gathered and reported. Much of it, of course, has no particular bearing upon this case. A great deal, and I might say, a preponderance of the reported material has to do with retrobulbar neuritis and iritis. Parsons, Fuchs and De Schweinitz agree that retrobulbar neuritis tends to spontaneous recovery. Iritis due to a specific infecting organism is more stubborn but is prone to clear up under treatment and to relapse. Sluder, in his work on "Nasal Neurology, Headaches and Eye Disorders," lays great stress on the relation between accessory sinus disease of the nose and eye disturbances. However, most of his observations have to do with ethmoiditis and sphenoiditis and not with infection of the maxillary sinuses as related to eye disease. This case is not one of sphenoiditis or of retrobulbar neuritis, but of retinal hemorrhage and massive hemorrhage into the vitreous.

Corneal ulcer, phlyctenular conjunctivitis and ulceration, often have no symptoms referable to the nose. It has been found that maxillary sinus disease is often the chief factor in promoting chronic eye disease, and ocular complications often are relieved only after treatment is directed to the relief of the antral infection.

Therson emphasizes eye complications in children, and states that retinitis may be caused by maxillary sinusitis. The offending pathogenic organism he found to be, most often, the diplococcus pneumoniæ. He declared that the roentgenogram is much more reliable as an aid to diagnosis than is transillumination.

McCabe reports hemorrhage from retinal vessels and hemorrhage into the vitreous humor caused by maxillary sinus disease.

Gunn states that rapid loss of vision in one eye points to sinus trouble and often to maxillary sinusitis, with or without subjective nasal symptoms.

Ocular sequelæ may result from lesions of any of the sinuses. Each sinus seems to have a fairly well established adherence to certain of these complications. Maxillary sinusitis may be responsible for edema of the lower lid, conjunctivitis, orbital cellulitis and abscess, optic neuritis and atrophy and, I might add, retinal hemorrhage, and hemorrhage into the vitreous humor.

One must remember and exclude infected or dead teeth as possible causes of eye diseases—e. g., glaucoma, iritis, retinitis and keratitis. Refractive error may play a part in centering the trouble in the eyes or their adnexa. In our experience the eye symptoms have disappeared entirely, and the vision has been greatly benefited by removal of all possible infection of the teeth. I may be classed as radical, since, in my opinion, no half way measures of treatment should ever be used. Extraction of the suspected teeth and curettement of the diseased bone have given the best results.

Reflex neurosis referable to the eyes may be expected when one recalls that the same sensory nerve supplies the nasal mucosa, the sinuses and the eye. In the study of focal infection it is frequently impressed upon us that disease of one part of the body, however latent or obscure, may be the cause of trouble in other parts, especially in the eye. In such cases the specialist often must be, for the time, a general practitioner and internist.

CONCLUSIONS.

1. Retrobulbar neuritis tends to clear up spontaneously, with or without treatment.
2. The great majority of cases which have been reported of eye disturbances due to sinus disease are not primarily due to maxillary sinusitis.
3. Maxillary sinusitis manifests itself in many ways other than by visual disturbances.
4. This case appears to be one of blindness with hemorrhage into the vitreous humor, due to focal infection originating in double chronic maxillary sinusitis.
5. In all cases of sudden retinal hemorrhage into the vitreous humor, maxillary antrum infection should be carefully investigated and excluded if possible.

6. Paranasal sinusitis is responsible for many cases of deeply seated eye lesions.

7. Eye disease and disturbance of vision may often be the result of maxillary sinusitis.

8. In many instances there is no direct complaint by the patient of nasal trouble, but careful and repeated examination will often reveal chronic suppuration of one or more of the sinuses.

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Abstracts of Current Articles

Dacryocystorhinostomy or Extirpation of the Lacrimal Sac.

R. Argañaraz (Buenos Aires), Arch. de Oftalm. de Buenos Aires, 3:475, 1928.

Practicing the Dupuy-Dutemps external operation in Segura's Clinic, Prof. Arganaraz points out the great difficulties of suturing H-shaped flaps of nasal mucosa to those of the lateral wall of the sac in so deep a wound. He has had to substitute removal of the sac in two cases because of severe hemorrhage, and has similarly given up use of chisels. Submucous injection of adrenalin-novocain helps with the operation but does not prevent severe after-hemorrhage. He considers the vaunted advantages of prevention of epiphora after such operations more theoretical than actual. F.

Serous Meningitis of Otitic Origin.

P. Moscardi (Rome), Riv. Oto-neur.-oft., 5:293, 1928.

Making ocular examinations of all meningitic cases in Ferreri's clinic, Moscardi recalls the resemblance of serous meningitis to serous exudates within the joint cavities connected with an inflamed long bone. Such serous processes may take place anywhere within the skull, rarely localized near the affected ear, but frequently attacking the choroid plexus and ventricular ependyma with alteration in or hypersecretion of cerebrospinal fluid. Such patches of defensive reaction are commonly aseptic. All cases of serous meningitis should have careful otologic study. Ocular findings include varying degrees of peripapillary edema of the retina, usually homolateral, with slight reduction of the field for green and slight enlargement of the blind spot upward and downward. Visual acuity was rarely affected. Occasional pareses of the sixth and third nerves were noted. Where postneuritic changes were visible, even if slight, the prognosis was notably worse. Eye symptoms were much helped after operation and after lumbar puncture. F.

Dacryoethmoiditis.

P. Moscardi (Rome), Riv. Oto-neur.-oft., 5:228, 1928.

Twenty out of one hundred and forty cases of dacryocystitis were found to have adjacent ethmoiditis. One case also had maxillary, and two both maxillary and frontal involvement in addition to the ethmoiditis. Inflammation transmitted to the lacrimal sac from without is rare; more frequent is an ethmoiditis discovered during removal of the sac, or giving rise to fistulization after such removal. Most frequent (75 per cent) are cases in which dacryocystitis and ethmoiditis coexist. All cases save one had the sac removed, with simultaneous transorbital exenteration of the anterior ethmoid cells. One dacryocystorhinostomy was done, with good functional result. Radiographs of the sinuses are recommended in all cases of dacryocystitis in which operation seems indicated. Greater intensity of the inflammatory process histologically suggests that most infections invade the ethmoid from the sac, perhaps with the help of trauma from probes or from operative procedures. F.

The Antivirus of Besredka for Local Treatment.

R. Vilar Fiol (Valencia), Revue de Lar., Otol., Rhin. (Moure), 49:495, 1928.

Recognizing that surgery is needed for suppurative foci and vaccine therapy as an immunizing agent against invasion of sound tissues, Vilar Fiol gives high rank to Besredka's antivirus, made from the appropriate local bacteria, especially staphylococci and streptococci, or of polyvalent strains of similar germs, for the treatment of wound cavities, fistulæ, osteomyelitis, postoperative neuralgias, sinus suppuration, external otitis, and even for pyorrhea. He claims very rapid local healing under the influence of these preparations, which are compatible both with surgery and with the use of vaccine therapy. Application is made by gauze packing or by injection. F.

Drainage of Double Frontal Sinusitis by Radical External Operation on One Side.

P. Caccialupi (Rome), Riv. Oto-neur.-oft., 5:271, 1928.

Accidental opening through the intersinuous septum afforded easy access for enlarging both nasofrontal ducts and is recom-

mended on subsequent trial as a time saving procedure, avoiding one external scar in cases of severe double frontal suppuration. F.

Osteomyelitis of the Superior Maxilla in Infants (So-called Sinusitis). (L'osteomyelite du maxillaire superieur dans le nourisson—la fausse sinusite.)

J. Terracol (Strasbourg), Arch. Int. Lar., 7:732, 1928.

The infantile antrum, 10 to 14 millimeters long and 3 or 4 wide, is crowded up toward the orbit by dental germs, and, according to Terracol, becomes infected more frequently after than during birth, generally by infection acquired during nursing. True osteomyelitic changes take place, causing unilateral nasal discharge, then swelling of the lids and cheek, which may be confused with dacryocystitis, gonorrheal ophthalmia or syphilitic osteitis. Fistulization may occur at the alveolar margin, the inner canthus, or through the hard palate. The rudimentary canine or premolar teeth will be found on incision of the labiogingival fold, lying bathed in pus, and the necrotic bone should be carefully curetted and, if possible, nasal drainage should be increased. Destruction of the spongy structure of the bone by excessive curettage is discouraged. In spite of the mortality of 30 to 50 per cent, due to serious systemic complications, the surviving cases present no facial or palatal asymmetry, save for the loss of teeth. F.

Therapeutic Indications in Laryngeal Tuberculosis. (Indications thérapeutiques dans la tuberculose laryngée.)

Caboche, H. (Paris), Arch. Int. Lar., 7:897, 1928.

This exhaustive report to the 1927 French congress of otolaryngologists brings out the importance of special hospital facilities for this class of tuberculous patients, and gives the results of a questionnaire answered by most of the prominent laryngologists of France and Italy.

Caboche considers general treatment by hygiene and diet, rest of the lung by pneumothorax, etc., and local rest through silence, to be an integral part of the treatment of laryngeal tuberculosis. All local treatment should be preceded by disinfectant laryngotracheal instillations. Radical local surgery (laryngectomy, laryngofissure) is never applicable. Epiglottectomy should be reserved to lesions of the free border of this

structure, and is best done by diathermic coagulation. Local application of lactic or trichloracetic acids is useful in ulcerative lesions of the true and false cords, and intratracheal use of cod liver oil is said to be desirable. Endolaryngeal surgery should be limited to removal of indolent granulations; the galvanocautery is to be preferred, especially in combination with heliotherapy or the quartz light. Results of radiotherapy are contradictory. Diathermy is not recommended except for readily accessible epiglottic lesions; the high frequency spark has so far not given convincing proof of its value. Recent use of benzyl-cinnamic ether by intramuscular injection has seemed to give general and local help. The methyl-antigen of Boquet and Négre is the most helpful of the antibody solutions thus far devised.

Caboche insists upon the curability of laryngeal tuberculosis, given early, methodical, persevering treatment by a laryngologist. Such cases demand proper hospitalization and the utilization of every modern resource. An excellent presentation of the Latin viewpoint, with recent European bibliography. F.

The Sphenopalatine System, Cervical Sympathetic and Ozena. (Bloc sphéno-palatin, sympathique cervical et ozène.)
Jung, Tagand & Chavanne (Lyon). Oto-Rhino-Lar. Internat., 12:449, 1928.

After four years of animal experiment in the Veterinary School at Lyon, these authors find no macroscopic or microscopic change in the nasal bones or mucosa, two to five months after painstaking removal of the sphenopalatine ganglion, the cervical sympathetic ganglion, or both. They incline with Escat to the theory of destruction of nervous and vascular elements in the mucosa itself, following the scarring of long continued local suppuration, rather than to trophic changes from distant nerve lesions. F.

Paralyses of the Recurrent in Esophageal Cancer. (Paralysies récurrentielles dans le cancer de l'oesophage.)
Sargnon, A. (Paris), Arch. Int. Lar., 7:1045, 1928.

Recurrent paralysis is the most frequent neurologic complication of esophageal cancer, may be bilateral, and should be carefully distinguished from that caused by aortic aneurysm because of the great danger from esophageal examination in

the latter type of case. Double recurrent palsy is found mainly in men, especially in cancer above the upper third of the thoracic esophagus. If unilateral, it is, as in aneurysm, usually of the left cord. Two types of bilateral palsy are distinguished: rapid, with extreme adduction, harsh voice and marked dyspnea, necessitating tracheotomy; and slow, aphonic, wherein one (left) cord is paralyzed first, then the other, allowing time for some separation of the cords, harsh breathing without dyspnea, and loss of phonation. As dysphagia may exist with aneurysm other distinguishing signs must always be looked for. Treatment, save for palliation by tracheotomy or gastrostomy, is futile. F.

Books Received

Fatalities from Local Anesthesia (Les Accidents Mortels de l'Anesthésic Locale).

By A. Klotz, M. D., Strasbourg. Paper. 12mo of 186 pages. Paris: Jouve & Cie., 1929.

This important thesis, inspired by Prof. Canuyt of Strasbourg, brings the exhaustive work of the A. M. A. Committee on Local Anesthetics and of its chairman, Dr. Emil Mayer, into relation with a mass of collected cases from all parts of Europe. Blegvad and more recently Canuyt have had the same trouble in securing reports of such cases as did Mayer. After a well-documented consideration of cocain and novocain, based on animal experiments, the author discusses in detail the toxicity of injected adrenalin. Synergistic action of potassium sulphate, ½ per cent, with novocain, 1/10 per cent, after Hoffmann and Kochmann, is recommended for injection, being equivalent to ½ per cent novocain with adrenalin as ordinarily used. Butyn, though almost as toxic as cocain, is thought by Klotz to possess slightly higher anesthetic power in its 5 per cent solution, and is less likely to produce psychic and cardiac disturbances.

Of 87 otolaryngologic fatalities tabulated and studied in detail, Klotz adds 18 to the 69 A. M. A. Committee cases cited. Including general and urologic surgery and stomatology, he recapitulates 151 cases. In general his findings repeat those emphasized by Mayer, Skillern and Sonnenschein.

Klotz agrees with Mayer as to the special danger of careless use of cocain paste. Nevertheless, he reports numerous cases of death from very weak solutions. The injection of adrenalin should be most carefully regulated. Death "by inhibition," in cases of endocrine imbalance, may be precipitated by very weak doses of novocain, cocain and/or adrenalin. The so-called "status lymphaticus" may be considered as indicative of such a predisposing neurovegetative imbalance; the so-called "thymic death" has up to the present failed to receive exact scientific corroboration.

Klotz rejects the air embolism theory. He reports 22 cases of excessive dosage, 9 cases due to direct injection into a vessel or into the meninges, 4 of excessive concentration. Twenty-four deaths showed no postmortem abnormalities whatever and may be classed as individually hypersensitive. He decries the use of local in combination with general anesthesia, especially with chloroform.

Fatigue, cachexia, alcoholism, advanced age, grave organic disease, call for great caution in local anesthesia. Ether is preferable to novocain in epileptics and tabetics. Adrenalin must be omitted or used with great caution in heart and goiter cases, diabetics, arteriosclerotics and pregnant women. Youth is not a contraindication to local anesthesia.

Following Canuyt and many American authorities, the semireclining or horizontal position is recommended during anesthesia and operation.

Treatment of syncopal seizures by artificial respiration, especially by insufflation of oxygen plus carbon dioxid, is highly recommended. Injection of lobelin as a respiratory stimulant is suggested. Convulsive seizures may be quieted by ether along with artificial respiration. If adrenalin has been injected it will be useless as a cardiac stimulant to use more. The usual cardiac stimulants should be available as emergency measures at all times.

An excellent bibliography completes this thesis, which is convincing evidence of the stimulating effect of Emil Mayer's work upon the younger generation of continental otolaryngologists. F.

A Short History of Medicine.
By Charles Singer, M. A., M. D., D. Litt., Fellow of the Royal College of Physicians of London. Cloth. 8vo of 368 pages with 142 illustrations. New York, Oxford University Press, 1928. Price, $3.00.

Although its title page describes this work as "introducing medical principles to students and nonmedical readers," it is by no means one of the now popular medical story books designed to enhance tea table conversation. On the contrary, the material is carefully and accurately prepared in sufficient detail to engage attention, and in a style to hold it.

To the specialist, focusing intently upon his subject, the study of the history of medicine should appeal peculiarly. To

him the world looks for advancement; his mind is on one thing; it is his privilege to concentrate upon a few problems. If he is honest and admits, at least to himself, that his progress has been slow, let him find solace in contemplation, for example, of the fact that although Aristotle, three centuries before Christ, "realized that the arteries are usually accompanied by veins," nevertheless nineteen hundred years elapsed before Harvey found out why.

But at the same time, let him give more than passing thought to a concluding paragraph in Dr. Singer's Epilogue: "The mass of scientific detail has always been beyond the power of one mind to grasp. But as we have traced Rational Medicine through its long course in antiquity and the Middle Ages to its debouchment on to our own time, we have found not only a more difficult but also a new situation. In approaching our own age we have found ever more difficulty in discussing Rational Medicine as a single channel of thought. It spreads into a delta, of which, though the many mouths may inosculate, yet the tendency seems to be for an ever wider divergence. This diffusion, induced by increased specialization, cannot go on for ever without defeating the very objects for which specialism was invented." P.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, October 1, 1928.

DR. WALTER H. THEOBALD IN THE CHAIR.

SCIENTIFIC PROGRAM.

DR. JOHN EDMUND MACKENTY, New York City (by invitation), read a paper on

"The Early Diagnosis and Treatment of Laryngeal Cancer."

(ABSTRACT.)

Malignant disease of the larynx occurs in 5 per cent of all malignant tumors and may be classified as 98 per cent carcinoma, 2 per cent sarcoma. Of the carcinomas 96 per cent are squamous celled and extremely malignant, varying considerably in degree. Of the less malignant types 2 per cent are basal celled, 1 per cent papillary and 1 per cent adenocarcinoma. From a surgical and prognostic standpoint the exact location of cancer in or about the larynx is of extreme importance. Clinically three classifications are recognized, based on the location and on the extent of the disease: extrinsic, intrinsic and borderline intrinsic. Extrinsic cancer rarely gives warning of its presence until well advanced. Intrinsic cancer in 90 per cent of the cases gives immediate notice of its presence in a hoarse or altered voice. Borderline intrinsic cancer is of the advanced intrinsic type, yet gives some hope of cure by surgery.

Persistent, progressive hoarseness in or after middle life may, and often does, mean laryngeal cancer, and calls for careful, conscientious and intelligent investigation by one competent to carry it out. A careful clinical history is invaluable in directing the search in the most probable direction. In looking into the larynx the following points must be noted: If a growth is in question, its position, size and characteristics are noted. If an inflammation is in question, its extent, location and peculiarities are observed. Pain is an important diagnostic

symptom. In the larynx tuberculosis is a painful disease. Syphilis is an anesthetic disease. Cancer is painful only when ulcerating, and then much less so than tuberculosis. Other laryngeal diseases are painful.

Early cancer must be suspected when the following conditions are present in a patient of cancer age: Progressive hoarseness; partial loss of mobility of cord on affected side, coming early in the disease; unilateral growth in anterior two-thirds of the larynx; syphilis and tuberculosis negative. Biopsy is permissible if the diagnosis cannot be made otherwise and the patient demands it. A negative biopsy should not stand against positive clinical findings. Even a positive finding should be questioned if not backed by clinical evidence. In all cases the opinion of two pathologists should be had. If cancer is the ultimate verdict the patient should be metabolically and otherwise prepared for operation, which should follow as closely upon the biopsy as possible.

In the author's personal experience since 1908 over 700 patients with cancer of the larynx have been studied. Of these, 80 per cent, in their inception, were intrinsic and curable. Treated surgically, 230, with a surgical mortality of under 3 per cent. Recurrences in incipient intrinsic cancer: After laryngectomy, 3 per cent; after thyrotomy, 35 per cent. Recurrences in intrinsic moderately advanced cancer, 25 per cent; recurrences in borderline cancer, 35 per cent; recurrences in extrinsic cancer, almost 100 per cent (the exception being two patients with early cancer of the epiglottis who have had no recurrence after nearly two years).

Cancer of the larynx is apparently on the increase and is now quite common in the thirties and even in the twenties, and not at all rare between seventy-five and eighty, although it was formerly limited almost entirely to the middle section of life. The author within eighteen months has removed the larynx from a man of twenty-nine and from a girl of twenty.

These statistics are a living argument for early diagnosis and radical treatment.

DISCUSSION.

Dr. Joseph C. Beck said that Glick, Sorensen and other men in Europe were among the first to recommend laryngec-

tomy, but the results were not very good. Then came the operation by Crile and that was very little better. Later came the question of X-ray and radium and burning out the larynx with the Percy cautery, and those patients did not do much better. They lived through the operation and they died of cancer as though they had not been operated upon, but since the perfection of the technic by Dr. Mackenty the results are very much better and there is a great deal more to be hoped for if the cases come early enough. Too many cases are not presented until the glands of the neck are enlarged. As a matter of fact, these should not be operated upon.

He emphasized some points in the technic employed by Dr. Mackenty. One technical point in particular is the corking of the trachea by means of a rubber tube. This keeps the secretions from running into the trachea, plugging the bronchi and setting up a septic bronchitis.

In the way of preoperative preparation it is important to see that the patient is a good risk. The anesthetic of choice is a synergistic analgesia. Dr. Beck has not used inhalation anesthesia. The use of the suction apparatus after operation to assist the patient so he will not have to cough is of great help. Feeding is carried on by means of the Rehfuss tube which the patient carries until the esophagus is healed. There should be day and night nurses to watch the patient carefully.

Dr. Mackenty emphasized the harm that comes from X-ray and radium treatment in these cases.

As to the age of the patient, last year Dr. Beck showed a patient, aged twenty-nine, whom he had operated upon for a cancer of the larynx. A recent report stated that the patient was working and in good condition. He had gained forty pounds in weight. He is occasionally asked if it is worth while to operate on a patient of sixty or seventy. If a patient is operated on for carcinoma of the larynx and lives six, seven or eight years the operation is worth while.

Dr. C. F. Yerger said he could not agree with the statement that cancer of the larynx offers the most favorable prognosis of cancer in any part of the body. He was of the opinion that cancer of the skin and mucocutaneous regions, as the lip, and especially the basal cell type of epitheliomata, offered a better prognosis; also, carcinoma of the cervix uteri, and possibly of

the breast. Regions of the body in which the cancer is visible early or those in which there are marked early symptoms offer a better prognosis because the patient seeks medical advice earlier.

The reason that cancer of the larynx affords a fairly good prognosis, much better than some of the deeper seated organs, is because the growth is generally situated on the vocal cord and produces hoarseness early in the course of the disease, thereby affording an early diagnosis and treatment. Other reasons are the favorable anatomic peculiarities of the larynx which may aid in the resistance to the development of a cancer, viz., the relatively sparse lymphatic circulation of the larynx, its protection by a natural barrier of cartilage, which cancer does not invade as freely as soft tissues, and because the larynx is very well isolated from its surrounding structures.

Cancer spreads and metastasizes by way of the lymphatics; hence, in cancer of any organ we must be familiar with its lymphatic circulation. Cuneo has shown that the lymphatics of the larynx may be divided into two groups, those above and those below the glottis. The lymphatics above the glottis empty into a lymphatic gland in the digastric region at the bifurcation of the carotid; the lymphatics below the glottis drain into the deep cervical lymphatic glands along the larynx and trachea, the supraclavicular glands and eventually may involve the mediastinal glands. Dr. Yerger saw a case of carcinoma of the larynx in which the only sign or symptom was an enlarged and hard lymph gland situated in the region of the bifurcation of the carotid. The diagnosis of carcinoma was made by biopsy and then an examination of the larynx disclosed a primary carcinoma of the larynx.

He emphasized the necessity of making both a direct and indirect laryngoscopy, also the difficulty of appraising correctly the extent of the carcinomatous involvement by laryngoscopic means, as exploration at operation generally shows that the carcinoma is twice as large as the laryngoscopic examination disclosed.

In his opinion, there is no endoscopic method of removing a cancer of the larynx completely and securely, and therefore preference should be given to laryngofissure.

Often there is a question as to whether a thyrotomy or a laryngectomy is indicated. This is a most important point to decide in the treatment. In some of the early cases it is not necessary to deprive the patient of his larynx. If the larynx can be retained without at the same time jeopardizing the life of the patient this would be the ideal method. Where the cancer is small and can be completely eradicated by excision, thyrotomy should be done, but the patient should be carefully watched and examined monthly thereafter for evidences of recurrence. If this occurs, a complete laryngectomy should be done.

He asked Dr. Mackenty whether he uses Broders' method of classification of the index of malignancy. Broders divides carcinoma into four groups, depending upon the degree of cell differentiation present. Group No. 1, where the tumor cells reach up to 25 per cent; No. 2, to 50 per cent; No. 3, to 75 per cent; No. 4, to 100 per cent. If the type is No. 1, and in some cases No. 2, the case is considered suitable for thyrotomy; if the type is No. 3 or No. 4, a conservative operation is contraindicated and a total laryngectomy is advisable.

Dr. G. W. Boot said he was glad to hear Dr. Mackenty say that he has found radium of little or no use, because that coincided with his experience. In his opinion, the ordinary patient is much worse off after radium has been used than if it is left out entirely.

He was rather surprised to hear Dr. Mackenty say that tuberculosis of the larynx always begins in one particular place. He was sure he had seen it begin in other places. He was also surprised to hear that cancer also begins in one place only. He had seen it begin simultaneously in wholly separate places. At a previous meeting he had told of a man who had two cancers and four precancerous lesions, which were probably cancers in reality, all starting at once. He also had a case of a man who had cancer, syphilis and tuberculosis of the larynx at the same time. Dr. Beck made one diagnosis, Dr. Friedberg the other, and he made the third, and at postmortem all three conditions were found.

He said that they have been getting remarkable results at the Cook County Hospital with diathermy in cancer of the

throat, that he considers it an agent that will supersede most of the agents now being used.

He was impressed with the statement of Dr. Mackenty about picking out good operative risks, patients who have syphilis, diabetes, cancer and a few other things, and he wondered what would constitute a poor risk.

Dr. Alfred Lewy said that much of Dr. Mackenty's success was due to his skill and resourcefulness in combating post-operative sequelæ. He asked for more detail in the methods employed by Dr. Mackenty, especially in the slow healing cases.

Dr. Walter H. Theobald asked what was the relative frequency of carcinoma of the larynx in the male and female.

Dr. John Edmund Mackenty, in closing, said he was not in position to discuss the two-stage operation referred to by Dr. Beck, for the reason that he had never adopted it. His early training in surgery made him feel that what one does surgically should be done at one time, if possible.

The highest hope in this work is to get the cases as early as possible. The earliest cases he has seen came from South America and Cuba. Spaniards are a hardy race and he has never had a Spaniard die following laryngectomy. The laryngologists in Cuba and South America seem more alert to the early manifestations of cancer.

Regarding the extrinsic operation, he said he had no success with it. In an extensive block operation of the neck, his patients all eventually died of cancer. Lately he has had two patients where only the top or anterior surface of the epiglottis was involved, and of these, one patient is alive two years and the other eighteen months after operation. Whether they will have a recurrence cannot be determined as yet. If a total laryngectomy is contemplated and a preliminary tracheotomy must be done, it is well to make the opening as high as possible so as to be able to remove the tracheotomy opening with the larynx. However, one should not go so high that he is in the cancer area.

Since safeguarding the patient from blood flowing into the trachea and larynx, he has had no case of pneumonia. Blood in the lungs at operation is equivalent to a foreign body. It forms a nidus for bacterial activity. Another thing that has helped is a special method of drainage. It has prevented me-

diastinitis. The progress made in metabolism has helped tremendously in getting these cases ready. The patient's blood chemistry should be carefully gone into and corrected if faulty. All patients over forty years of age should be digitalized. After operation the patient is fed through a tube which is placed into the esophagus about six inches below the larynx. The whole operation can be done under local anesthesia if desired, but it is too severe a shock to the nervous system. The patient's strength is better conserved by a combined anesthesia (scopolamin 1/200, morphin ¼, regional novocain 1 per cent to ½ of 1 per cent and ether). About one-half the operation is done under local anesthesia.

He believed there is a specific toxemia with carcinoma, and when cancer is removed the patient takes on a new lease of life. He did not think it could be said that carcinoma is ever cured. It is like syphilis and tuberculosis—arrested.

Answering Dr. Yerger's question regarding early carcinoma in other parts of the body, he said that while he was not acquainted with general surgery, he understood that surgeons were obtaining only 50 per cent of cures in cancer of the breast, and the results in cancer of the larynx were better than that. Cancer of the cervix, if taken early and if an extensive operation is done, probably offers as good or maybe better results. Skin cancer does not come under the same category, because it is not so malignant. Cancers of the mouth are very malignant unless taken very early.

The recurrences have been in the neck glands in all of the cases he has seen. The region about the carotid angle is most likely to be involved. He has never seen a case of recurrence in the mediastinum. This is probably due to the fact that there is a separate set of lymphatics for the neck and for the mediastinum. So far as he knows, these do not intercommunicate.

Dr. Yerger was correct about estimating the amount of cancer in the larynx. It is very difficult to tell how large a cancer is by looking at it from above. When the larynx is opened it is usually three times as extensive as it seemed.

In one of the cases reported by Dr. Mackenty, when Jackson made the biopsy, there was no cancer. The patient developed shortly afterward a fulminating cancer. He had, what is now being more and more recognized, a precancerous condition.

As to thyrotomy and laryngectomy, he is not against doing thyrotomy when it is indicated. He has not had the glorious results reported by the men abroad with thyrotomy. He has had 35 per cent recurrence, but he does not get the early cases. For thyrotomy, the Thompson method is probably the better. Years ago he did a hemilaryngectomy in several cases, but the mortality was greater than in total extirpation. Thompson's operation lies somewhere between a thyrotomy and a hemilaryngectomy. He removes the thyroid cartilage over the diseased area. This has only one disadvantage: if there is a recurrence it will invade the neck at once, because there is no cartilaginous barrier to prevent or retard it.

To a certain extent he believes in Broders' method of classification, though he had seen cases where it did not hold, where the malignancy was very pronounced in spite of the fact that the cells were very few.

He was very glad to hear Dr. Boot say what he did about radium. Jackson once believed in radium, but he now feels much as the speaker and Dr. Boot do. Radium is good in certain things, such as skin cancer, but in this particular condition it is not.

He did not intend to convey the idea that tuberculosis always occurs in one place. He was trying to get some kind of classification on the question of diagnosis. Tuberculosis is very liable to invade the posterior part of the larynx. Tuberculosis may begin anywhere in the larynx, the same as any tumor. It may begin in the epiglottis and the epiglottic folds, but when it involves the inside of the larynx it is very liable to attack the posterior commissure. He did not believe that tuberculosis of the larynx occurred unless there was a precedent tuberculosis of the lung, either latent or active. That should be decided by expert examination of the lungs. It is not always found on ordinary examination.

Dr. Lewy asked about the slow healing cases. The severe cases are due to radium. Here it is often impossible to effect a closure. The whole wound may fall apart, leaving a large dehiscence or only a fistula may remain.

He uses a T incision. It is important to get the closure at the angel of the T in order to avoid an open hypopharynx. It may break down on either side and heal up without trouble,

but if this bridge breaks down, a fistula is sure to result and a plastic will be necessary. He closes with a mattress suture, which gives a good line of contact. The closure is simple. When the wound breaks down it is necessary to wait until every bit of infection is gone before attempting closure. This can be done under local anesthetic. The tissues are split and the inner flap inverted with mattress sutures and the outer everted with the same stitch. When suturing is completed there should be a ridge of tissue on the line of union.

Answering Dr. Theobald's question, he said that cancer of the larynx in the female is very rare, not more than 5 or 6 per cent. Thompson of London has observed about the same percentage, but Dr. Mackenty believed that cancer in the female is becoming more frequent. He has seen five in the last four years: one in a patient aged 20, two in patients aged 26, one in a patient in the early thirties, and one in a woman aged 58. It seemed to him that laryngeal cancer in the female occurs at an earlier age than in the male.

Contributors to This Number

J. S. FRASER is the head of the Ear and Throat Department of the Royal Infirmary of Edinburgh. His article in this issue is the highly instructive resumé of the work of this Ear and Throat Clinic for 1927. Until January of this year, Dr. Fraser was co-editor of the Journal of Laryngology and Otology. In 1923, he was the guest of the American Laryngological Association and the American Laryngological, Rhinological and Otological Society, at which time he presented his exhaustive researches on the pathology of labyrinthitis.

GEORGES PORTMANN is Clinical Professor of Otorhinolaryngology at the University of Bordeaux. He is the author of several books, including a work on local anesthesia, reviewed in the September number of the ANNALS, and one on pharmacology for laryngologists. He has been the guest of various otolaryngologic societies in this country on several occasions.

ROBERT SONNENSCHEIN, of Chicago, is Assistant Clinical Professor of Otolaryngology at Rush Medical College and Professor of Otolarynogology in the Postgraduate Medical School. He is best known for his work on the standardization of tuning forks and hearing tests, and has been for several years Chairman of the American Academy's committee on hearing tests.

ARTHUR SCHÜLLER, author of the very complete and informative article, *"Roentgenological Study of Deformities of the Nasopharynx,"* is head of the Clinic for Nervous Diseases, Franz Joseph Ambulatorium, Vienna. Professor Schüller recently completed an extended tour of the clinics of this country and is in keen sympathy with the American viewpoint.

EUGENE R. LEWIS has a large affiliation with the hospitals of Los Angeles. His contributions have been chiefly on otologic subjects, notably those on the nature of nystagmus and on differential tests for stapes fixation.

DAN MACKENZIE, who contributes the article *"Treatment of Cancer of the Pharynx, Larynx and Esophagus by Surgical Diathermy,"* requires no introduction to the laryngologist. He

is the author of the well-known textbook and of several other works, including a fascinating little volume entitled "*Aromatics and the Soul.*"

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CONTENTS—Continued From Second Cover Page.

XV.—Anatomy, Physiology and Pathology of the Lacrimal Apparatus, More Especially the Lacrimal Sac and Duct. By Harry Vanderbilt Würdemann, M. D., Seattle....199

XVI.—Retropharyngeal Abscess. By Roy A. Barlow, M. D., Madison, Wis....205

XVII.—Complications of Otitis Media and Mastoiditis. By Edward H. Campbell, M. D., Philadelphia....209

XVIII.—Infantile Mastoiditis With Gastrointestinal Symptoms: Report of Two Cases. By James B. Costen, M. D., St. Louis....242

XIX.—Noncoalescent Mastoiditis With a Report of Four Cases. By Sam B. Marks, M. D., Lexington, Ky....251

XX.—The Nature of Nystagmus and Dizziness Occurring in Certain Positions of the Head and in Movements Other Than Those Around the Long Axis of the Body. By Ignatz Sommer, M. D., Vienna, Austria, and Joseph C. Yaskin, M. D., Philadelphia....260

XXI.—A Positive Symptom of Mastoiditis. By Murray Berger, M. D., New York....275

XXII.—Chronic Laryngeal Stenosis Complicating Laryngeal Diphtheria. By R. J. Payne, M. D., St. Louis....276

XXIII.—Toxic Otitis. By J. J. Hompes, M. D., Lincoln, Neb. 280

XXIV.—Unilateral Blindness: Recovery After Sinus Operation. By J. T. Dowling, M. D., Seattle....288

Abstracts of Current Articles....293

Books Received....298

Society Proceedings....301

Chicago Laryngological and Otological Society. Meeting of Monday, October 1, 1928—"The Early Diagnosis and Treatment of Laryngeal Cancer."....301

Contributors to This Number....310